U.S. DEPARTMENT OF COMMERCE
Patent and Trademark Office

SEARCH REQUEST FORM

5-653

Requestor's Name: Cook 2807 Serial Number: 09/009213

Date: 5/19/98 Phone: 308 4724 Art Unit: 1614

Search Topic:
Please write a detailed statement of search topic. Describe specifically as possible the subject matter to be searched. Define any terms that may have a special meaning. Give examples or relevent citations, authors, keywords, etc., if known. For sequences, please attach a copy of the sequence. You may include a copy of the broadest and/or most relevent claim(s).

Please search concept of reducing hair growth and increasing hair growth using same compounds on different parts of body. (See attached from specification and claims using UGT, ST and other compounds that convert androgens to less active metabolites)

Specific UGTs + ST's are claimed in dependent claims.

Inventor is Peter Stygynski

Thanks
Rebecca

60
21

STAFF USE ONLY

Date completed: 5/29/98
Searcher: K. Fuller
Terminal time: 180
Elapsed time:
CPU time:
Total time: 210
Number of Searches:
Number of Databases:

Search Site
- STIC
- ✓ CM-1
- Pre-S

Type of Search
- N.A. Sequence
- A.A. Sequence
- Structure
- ✓ Bibliographic

Vendors
- IG
- ✓ STN
- Dialog
- APS
- Geninfo
- SDC
- DARC/Questel
- Other

PTO-1590 (9-90)

=> FILE HCAPLUS

FILE 'HCAPLUS' ENTERED AT 11:11:29 ON 29 MAY 1998
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FILE COVERS 1967 - 29 May 1998 VOL 128 ISS 22
FILE LAST UPDATED: 29 May 1998 (980529/ED)

This file contains CAS Registry Numbers for easy and accurate substance identification.

This file now supports REG1stRY for direct browsing and searching of all non-structural data from the REGISTRY file. Enter HELP FIRST for more information.

=> D QUE L68

L25 (1)SEA FILE=REGISTRY ABB=ON ETHOXYQUIN/CN
L26 (1)SEA FILE=REGISTRY ABB=ON "5,7-DIHYDROXY-4'-METHOXYFLAVONE"/CN
L27 (1)SEA FILE=REGISTRY ABB=ON BUTYLHYDROXYANISOLE/CN
L28 (1)SEA FILE=REGISTRY ABB=ON PHENOBARBITAL/CN
L29 (1)SEA FILE=REGISTRY ABB=ON NARINGENIN/CN
L30 (3)SEA FILE=REGISTRY ABB=ON BUTYLHYDROXY(L)TOLUENE
L31 (1)SEA FILE=REGISTRY ABB=ON "PHENOL, 2-(1,1-DIMETHYLETHYL)-3-METHYL-"/CN
L32 (1)SEA FILE=REGISTRY ABB=ON "PHENOL, 2-(1,1-DIMETHYLETHYL)-4-METHYL-"/CN
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L34 (1)SEA FILE=REGISTRY ABB=ON "PHENOL, 3-(1,1-DIMETHYLETHYL)-5-METHYL-"/CN
L35 (1)SEA FILE=REGISTRY ABB=ON "PHENOL, 3-(1,1-DIMETHYLETHYL)-4-METHYL-"/CN
L36 (1)SEA FILE=REGISTRY ABB=ON "PHENOL, 3-(1,1-DIMETHYLETHYL)-2-METHYL-"/CN
L37 (1)SEA FILE=REGISTRY ABB=ON "PHENOL, 4-(1,1-DIMETHYLETHYL)-2-METHYL-"/CN
L38 (1)SEA FILE=REGISTRY ABB=ON "PHENOL, 4-(1,1-DIMETHYLETHYL)-3-METHYL-"/CN
L39 (1)SEA FILE=REGISTRY ABB=ON FLAVONE/CN
L40 (1)SEA FILE=REGISTRY ABB=ON TIOCONAZOLE/CN
L41 (1)SEA FILE=REGISTRY ABB=ON "TRANS-1,2-BIS(2-PYRIDYL)ETHYLENE"/CN
L42 (1)SEA FILE=REGISTRY ABB=ON "4',7-ISOFLAVANDIOL"/CN
L43 (1)SEA FILE=REGISTRY ABB=ON GALANGIN/CN
L44 (1)SEA FILE=REGISTRY ABB=ON "7-HYDROXY-4'-METHOXYISOFLAVONE"/CN
L45 (1)SEA FILE=REGISTRY ABB=ON DAIDZEIN/CN
L46 (19)SEA FILE=REGISTRY ABB=ON (L25 OR L26 OR L27 OR L28 OR L29 OR L30 OR L31 OR L32 OR L33 OR L34 OR L35 OR L36 OR L37 OR L38 OR L39 OR L40 OR L41)
L47 (4)SEA FILE=REGISTRY ABB=ON (L42 OR L43 OR L44 OR L45)
L48 (23)SEA FILE=REGISTRY ABB=ON L46 OR L47
L49 (18484)SEA FILE=HCAPLUS ABB=ON L48
L50 (57)SEA FILE=HCAPLUS ABB=ON L49 AND HAIR
L51 (13)SEA FILE=HCAPLUS ABB=ON L50 AND GROW?

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L52 (        19)SEA FILE=HCAPLUS ABB=ON  L49 AND (HIRSUT? OR ALOPEC?)
L53 (         6)SEA FILE=HCAPLUS ABB=ON  L52 AND GROW?
L54 (        13)SEA FILE=HCAPLUS ABB=ON  L51 OR L53
L55 (      1418)SEA FILE=HCAPLUS ABB=ON  ?GLUCURONOSYLTRANSFERAS?
L56 (      2051)SEA FILE=HCAPLUS ABB=ON  ?SULFOTRANSFERAS?
L57 (        15)SEA FILE=HCAPLUS ABB=ON  (L55 OR L56) AND HAIR
L58 (        13)SEA FILE=HCAPLUS ABB=ON  L57 AND GROW?
L59 (     52109)SEA FILE=HCAPLUS ABB=ON  ?ANDROGEN? OR ?TESTOSTERON?
L60 (       235)SEA FILE=HCAPLUS ABB=ON  L59 AND (HAIR(S)GROW?)
L61 (        41)SEA FILE=HCAPLUS ABB=ON  L60 AND PHARMACE?/SC,SX,AB,BI
L62 (        10)SEA FILE=HCAPLUS ABB=ON  L61 AND STIMULAT? AND INHIBIT?
L63 (         0)SEA FILE=HCAPLUS ABB=ON  L61 AND MODULAT?
L64          35 SEA FILE=HCAPLUS ABB=ON  L54 OR L58 OR L62 OR L63
L65           1 SEA FILE=REGISTRY ABB=ON  "5,4'-DIHYDROXY-7-METHOXYISOFLA
                VONE"/CN
L66          59 SEA FILE=HCAPLUS ABB=ON  L65
L67           0 SEA FILE=HCAPLUS ABB=ON  L66 AND HAIR AND GROW?
L68          35 SEA FILE=HCAPLUS ABB=ON  L64 OR L67

=> FILE BIOSIS

FILE 'BIOSIS' ENTERED AT 11:11:47 ON 29 MAY 1998
COPYRIGHT (C) 1998 BIOSIS(R)

FILE COVERS 1969 TO DATE.
CAS REGISTRY NUMBERS AND CHEMICAL NAMES (CNs) PRESENT
FROM JANUARY 1969 TO DATE.

RECORDS LAST ADDED: 20 May 1998 (980520/ED)
CAS REGISTRY NUMBERS (R) LAST ADDED: 20 May 1998 (980520/UP)


=> D QUE L89

L25 (         1)SEA FILE=REGISTRY ABB=ON  ETHOXYQUIN/CN
L26 (         1)SEA FILE=REGISTRY ABB=ON  "5,7-DIHYDROXY-4'-METHOXYFLAVON
                E"/CN
L27 (         1)SEA FILE=REGISTRY ABB=ON  BUTYLHYDROXYANISOLE/CN
L28 (         1)SEA FILE=REGISTRY ABB=ON  PHENOBARBITAL/CN
L29 (         1)SEA FILE=REGISTRY ABB=ON  NARINGENIN/CN
L30 (         3)SEA FILE=REGISTRY ABB=ON  BUTYLHYDROXY(L)TOLUENE
L31 (         1)SEA FILE=REGISTRY ABB=ON  "PHENOL, 2-(1,1-DIMETHYLETHYL)-
                3-METHYL-"/CN
L32 (         1)SEA FILE=REGISTRY ABB=ON  "PHENOL, 2-(1,1-DIMETHYLETHYL)-
                4-METHYL-"/CN
L33 (         1)SEA FILE=REGISTRY ABB=ON  "PHENOL, 2-(1,1-DIMETHYLETHYL)-
                5-METHYL-"/CN
L34 (         1)SEA FILE=REGISTRY ABB=ON  "PHENOL, 3-(1,1-DIMETHYLETHYL)-
                5-METHYL-"/CN
L35 (         1)SEA FILE=REGISTRY ABB=ON  "PHENOL, 3-(1,1-DIMETHYLETHYL)-
                4-METHYL-"/CN
L36 (         1)SEA FILE=REGISTRY ABB=ON  "PHENOL, 3-(1,1-DIMETHYLETHYL)-
                2-METHYL-"/CN
L37 (         1)SEA FILE=REGISTRY ABB=ON  "PHENOL, 4-(1,1-DIMETHYLETHYL)-
                2-METHYL-"/CN
L38 (         1)SEA FILE=REGISTRY ABB=ON  "PHENOL, 4-(1,1-DIMETHYLETHYL)-
                3-METHYL-"/CN
L39 (         1)SEA FILE=REGISTRY ABB=ON  FLAVONE/CN
L40 (         1)SEA FILE=REGISTRY ABB=ON  TIOCONAZOLE/CN
L41 (         1)SEA FILE=REGISTRY ABB=ON  "TRANS-1,2-BIS(2-PYRIDYL)ETHYLE
                NE"/CN
L42 (         1)SEA FILE=REGISTRY ABB=ON  "4',7-ISOFLAVANDIOL"/CN
L43 (         1)SEA FILE=REGISTRY ABB=ON  GALANGIN/CN
L44 (         1)SEA FILE=REGISTRY ABB=ON  "7-HYDROXY-4'-METHOXYISOFLAVONE
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               "/CN
L45 (          1)SEA FILE=REGISTRY ABB=ON  DAIDZEIN/CN
L46 (         19)SEA FILE=REGISTRY ABB=ON  (L25 OR L26 OR L27 OR L28 OR L2
                  9 OR L30 OR L31 OR L32 OR L33 OR L34 OR L35 OR L36 OR L37
                   OR L38 OR L39 OR L40 OR L41)
L47 (          4)SEA FILE=REGISTRY ABB=ON  (L42 OR L43 OR L44 OR L45)
L48 (         23)SEA FILE=REGISTRY ABB=ON  L46 OR L47
L65            1 SEA FILE=REGISTRY ABB=ON  "5,4'-DIHYDROXY-7-METHOXYISOFLA
                  VONE"/CN
L69         8348 SEA FILE=BIOSIS ABB=ON  L48 OR L65
L70           18 SEA FILE=BIOSIS ABB=ON  L69 AND HAIR
L72            6 SEA FILE=BIOSIS ABB=ON  L70 AND 86215/BC
L73            2 SEA FILE=BIOSIS ABB=ON  L70 AND  86375/BC
L74            8 SEA FILE=BIOSIS ABB=ON  L72 OR L73
L75         1418 SEA FILE=BIOSIS ABB=ON  GLUCURONOSYLTRANSFERASE? OR UGT O
                  R SULFOTRANFERAS?
L76            2 SEA FILE=BIOSIS ABB=ON  L75 AND HAIR
L77          698 SEA FILE=BIOSIS ABB=ON  (ANDROGEN? OR TESTOSTERON? ) AND
                  HAIR
L78          311 SEA FILE=BIOSIS ABB=ON  L77 AND GROW?
L79           13 SEA FILE=BIOSIS ABB=ON  L78 AND STIMULAT? AND INHIBIT?
L80          171 SEA FILE=BIOSIS ABB=ON  L78 AND 220?/CC
L81            1 SEA FILE=BIOSIS ABB=ON  L80 AND MODULAT?
L82            7 SEA FILE=BIOSIS ABB=ON  L79 AND L80
L83          114 SEA FILE=BIOSIS ABB=ON  L80 AND (THERAP? OR TREAT?)
L84          105 SEA FILE=BIOSIS ABB=ON  L83 AND 86215/BC
L85         1143 SEA FILE=BIOSIS ABB=ON  HAIR(W)GROWTH
L86           76 SEA FILE=BIOSIS ABB=ON  L84 AND L85
L87            3 SEA FILE=BIOSIS ABB=ON  L79 AND L86
L88           14 SEA FILE=BIOSIS ABB=ON  L86 AND INCREAS? AND (REDUC? OR I
                  NHIBIT?)
L89           30 SEA FILE=BIOSIS ABB=ON  L74 OR L76 OR L81 OR L82 OR L87 O
                  R L88

=> FILE WPIDS

FILE 'WPIDS' ENTERED AT 11:12:02 ON 29 MAY 1998
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FILE LAST UPDATED: 28 MAY 1998              <19980528/UP>
>>>UPDATE WEEKS:
MOST RECENT DERWENT WEEK            199821   <199821/DW>
DERWENT WEEK FOR CHEMICAL CODING:   199816
DERWENT WEEK FOR POLYMER INDEXING:  199818
DERWENT WORLD PATENTS INDEX SUBSCRIBER FILE, COVERS 1963 TO DATE
>>> D COST AND SET NOTICE DO NOT REFLECT SUBSCRIBER DISCOUNTS -
                                  SEE HELP COST FOR DETAILS <<<
>>> MEXICO NOW COVERED  - SEE NEWS <<<

=> D QUE L106

L90         1745 SEA FILE=WPIDS ABB=ON  HAIR(4A)GROW?
L91          193 SEA FILE=WPIDS ABB=ON  L90 AND (STIMULAT? OR INCREAS?) AN
                 D (INHIBIT? OR DECREAS? OR REDUC?)
L94           27 SEA FILE=WPIDS ABB=ON  L91 AND (?ANDROGEN? OR ?TESTOSTERO
                 N?)
L95           27 SEA FILE=WPIDS ABB=ON  L94 AND A61K0?/IC
L96           41 SEA FILE=WPIDS ABB=ON  ETHOXYQUIN OR R00581/DCN OR 581/DR
                 N
L97        98597 SEA FILE=WPIDS ABB=ON  PHENOBARBITAL OR R00005/DCN OR 5/D
                 RN
L98           37 SEA FILE=WPIDS ABB=ON  NARINGENIN OR R03812/DCN OR 3812/D
                 RN
L99          463 SEA FILE=WPIDS ABB=ON  FLAVONE OR 3811/DRN OR R03811/DCN
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L100          34 SEA FILE=WPIDS ABB=ON  TIOCONAZOL? OR R09317/DCN OR 9317/
                 DRN
L101         138 SEA FILE=WPIDS ABB=ON  ?ISOFLAVONE? OR ?ISOFLAVANDIOL?
L102          13 SEA FILE=WPIDS ABB=ON  GALANGIN OR R08508/DCN OR 8508/DRN

L103          29 SEA FILE=WPIDS ABB=ON  DAIDZEIN
L104       99242 SEA FILE=WPIDS ABB=ON  (L96 OR L97 OR L98 OR L99 OR L100
                 OR L101 OR L102 OR L103)
L105           5 SEA FILE=WPIDS ABB=ON  L90 AND L104
L106          32 SEA FILE=WPIDS ABB=ON  L95 OR L105

=> FILE MEDLINE

FILE 'MEDLINE' ENTERED AT 11:12:18 ON 29 MAY 1998

 FILE LAST UPDATED: 20 MAY 1998 (19980520/UP). FILE COVERS 1966 TO DATE.

 THE MEDLINE FILE WAS RELOADED FEBRUARY 15, 1998, TO REFLECT THE ANNUAL
 MESH (MEDICAL SUBJECT HEADING) CHANGES. ENTER HELP RLOAD FOR DETAILS.

 THIS FILE CONTAINS CAS REGISTRY NUMBERS FOR EASY AND ACCURATE
 SUBSTANCE IDENTIFICATION.

=> D QUE L129

L25 (          1)SEA FILE=REGISTRY ABB=ON  ETHOXYQUIN/CN
L26 (          1)SEA FILE=REGISTRY ABB=ON  "5,7-DIHYDROXY-4'-METHOXYFLAVON
                 E"/CN
L27 (          1)SEA FILE=REGISTRY ABB=ON  BUTYLHYDROXYANISOLE/CN
L28 (          1)SEA FILE=REGISTRY ABB=ON  PHENOBARBITAL/CN
L29 (          1)SEA FILE=REGISTRY ABB=ON  NARINGENIN/CN
L30 (          3)SEA FILE=REGISTRY ABB=ON  BUTYLHYDROXY(L)TOLUENE
L31 (          1)SEA FILE=REGISTRY ABB=ON  "PHENOL, 2-(1,1-DIMETHYLETHYL)-
                 3-METHYL-"/CN
L32 (          1)SEA FILE=REGISTRY ABB=ON  "PHENOL, 2-(1,1-DIMETHYLETHYL)-
                 4-METHYL-"/CN
L33 (          1)SEA FILE=REGISTRY ABB=ON  "PHENOL, 2-(1,1-DIMETHYLETHYL)-
                 5-METHYL-"/CN
L34 (          1)SEA FILE=REGISTRY ABB=ON  "PHENOL, 3-(1,1-DIMETHYLETHYL)-
                 5-METHYL-"/CN
L35 (          1)SEA FILE=REGISTRY ABB=ON  "PHENOL, 3-(1,1-DIMETHYLETHYL)-
                 4-METHYL-"/CN
L36 (          1)SEA FILE=REGISTRY ABB=ON  "PHENOL, 3-(1,1-DIMETHYLETHYL)-
                 2-METHYL-"/CN
L37 (          1)SEA FILE=REGISTRY ABB=ON  "PHENOL, 4-(1,1-DIMETHYLETHYL)-
                 2-METHYL-"/CN
L38 (          1)SEA FILE=REGISTRY ABB=ON  "PHENOL, 4-(1,1-DIMETHYLETHYL)-
                 3-METHYL-"/CN
L39 (          1)SEA FILE=REGISTRY ABB=ON  FLAVONE/CN
L40 (          1)SEA FILE=REGISTRY ABB=ON  TIOCONAZOLE/CN
L41 (          1)SEA FILE=REGISTRY ABB=ON  "TRANS-1,2-BIS(2-PYRIDYL)ETHYLE
                 NE"/CN
L42 (          1)SEA FILE=REGISTRY ABB=ON  "4',7-ISOFLAVANDIOL"/CN
L43 (          1)SEA FILE=REGISTRY ABB=ON  GALANGIN/CN
L44 (          1)SEA FILE=REGISTRY ABB=ON  "7-HYDROXY-4'-METHOXYISOFLAVONE
                 "/CN
L45 (          1)SEA FILE=REGISTRY ABB=ON  DAIDZEIN/CN
L46 (         19)SEA FILE=REGISTRY ABB=ON  (L25 OR L26 OR L27 OR L28 OR L2
                 9 OR L30 OR L31 OR L32 OR L33 OR L34 OR L35 OR L36 OR L37
                  OR L38 OR L39 OR L40 OR L41)
L47 (          4)SEA FILE=REGISTRY ABB=ON  (L42 OR L43 OR L44 OR L45)
L48 (         23)SEA FILE=REGISTRY ABB=ON  L46 OR L47
L65            1 SEA FILE=REGISTRY ABB=ON  "5,4'-DIHYDROXY-7-METHOXYISOFLA
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                VONE"/CN
L107         7996 SEA FILE=MEDLINE ABB=ON  L48 OR L65
L108        12344 SEA FILE=MEDLINE ABB=ON  HAIR+NT/CT
L109            8 SEA FILE=MEDLINE ABB=ON  L107 AND L108
L110            1 SEA FILE=MEDLINE ABB=ON  L107 AND HAIR(4A)GROW?
L114         2290 SEA FILE=MEDLINE ABB=ON  HIRSUTISM+NT/CT
L115            0 SEA FILE=MEDLINE ABB=ON  L107 AND L114
L116         5059 SEA FILE=MEDLINE ABB=ON  ALOPECIA+NT/CT
L117            0 SEA FILE=MEDLINE ABB=ON  L107 AND L116
L118         1233 SEA FILE=MEDLINE ABB=ON  L108(L)GD/CT
L119          370 SEA FILE=MEDLINE ABB=ON  (L118 OR L114) AND L116
L120          166 SEA FILE=MEDLINE ABB=ON  L119 AND DT/CT
L121          158 SEA FILE=MEDLINE ABB=ON  L120/HUMAN
L122          128 SEA FILE=MEDLINE ABB=ON  L121 AND TU/CT
L123         2419 SEA FILE=MEDLINE ABB=ON  GLUCURONOSYLTRANSFERASE? OR UGT
                  OR SULFOTRANFERAS?
L124            0 SEA FILE=MEDLINE ABB=ON  L122 AND L123
L125            0 SEA FILE=MEDLINE ABB=ON  L119 AND L123
L126            1 SEA FILE=MEDLINE ABB=ON  L108 AND L123
L127         2152 SEA FILE=MEDLINE ABB=ON  GLUCURONOSYLTRANSFERASE+NT/CT
L128            1 SEA FILE=MEDLINE ABB=ON  L127 AND  (L108 OR L114 OR L116)

L129            9 SEA FILE=MEDLINE ABB=ON  L109 OR L110 OR L115 OR L117 OR
                  L124 OR  L125 OR L126 OR L128

=> DUP REM L68 L89 L106 L129

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FILE 'BIOSIS' ENTERED AT 11:12:45 ON 29 MAY 1998
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FILE 'WPIDS' ENTERED AT 11:12:45 ON 29 MAY 1998
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FILE 'MEDLINE' ENTERED AT 11:12:45 ON 29 MAY 1998
PROCESSING COMPLETED FOR L68
PROCESSING COMPLETED FOR L89
PROCESSING COMPLETED FOR L106
PROCESSING COMPLETED FOR L129
L130           97 DUP REM L68 L89 L106 L129 (9 DUPLICATES REMOVED)

=> D L130 ALL 1-97


L130 ANSWER 1 OF 97  HCAPLUS  COPYRIGHT 1998 ACS
AN   1998:76215  HCAPLUS
DN   128:196471
TI   Antiandrogens containing jasmone, etc., and their uses for
     hair preparations
IN   Seiki, Hitoshi; Okano, Yuri; Torii, Hirosuke
PA   NOEVIR Co., Ltd., Japan
SO   Jpn. Kokai Tokkyo Koho, 7 pp.
     CODEN: JKXXAF
PI   JP 10029935 A2  980203  Heisei
AI   JP 96-203045  960712
DT   Patent
LA   Japanese
IC   ICM  A61K031-12
     ICS  A61K031-12; A61K007-00; A61K007-06; A61K031-215; A61K035-78
CC   62-3 (Essential Oils and Cosmetics)
```

Section cross-reference(s): 1, **63**

AB The **antiandrogens, hair growth** stimulants, and **hair** prepns. contain .gtoreq.1 selected from cis-jasmone (I), Me dihydroisojasmonate, Me dihydrojasmonate, and dihydrojasmone. The **antiandrogens** are useful for treatment of prostatic hypertrophy, prostatic cancer, early manifestation of secondary sexual characters in boys, psoriasis, seborrhea, etc. I **inhibited testosterone-stimulated** proliferation of **androgen**-dependent mouse spontaneous mammary cancer cell SC-3. A hair treatment contg. I was also prepd.

ST jasmone **antiandrogen** drug **hair growth** stimulant; dihydroisojasmonate **antiandrogen** drug **hair growth** stimulant; dihydrojasmonate **antiandrogen** drug **hair growth** stimulant; dihydrojasmone **antiandrogen** drug **hair growth** stimulant; **androgen** dependent disease **inhibitor** jasmone dihydrojasmonate

IT **Hair growth** stimulants
Hair preparations
(**antiandrogen** effect of jasmone, Me dihydro(iso)jasmonate, and dihydrojasmone and their application to drugs and **hair** prepns.)

IT **Antiandrogens**
RL: BAC (Biological activity or effector, except adverse); BUU (Biological use, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**antiandrogen** effect of jasmone, Me dihydro(iso)jasmonate, and dihydrojasmone and their application to drugs and hair prepns.)

IT **Androgens**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(disease dependent on; **antiandrogen** effect of jasmone, Me dihydro(iso)jasmonate, and dihydrojasmone and their application to drugs and hair prepns.)

IT 488-10-8, cis-Jasmone 1128-08-1, Dihydrojasmone 2630-39-9, Methyl dihydrojasmonate 39647-11-5
RL: BAC (Biological activity or effector, except adverse); BUU (Biological use, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**antiandrogen** effect of jasmone, Me dihydro(iso)jasmonate, and dihydrojasmone and their application to drugs and hair prepns.)

L130 ANSWER 2 OF 97 HCAPLUS COPYRIGHT 1998 ACS DUPLICATE 1

AN 1998:164992 HCAPLUS

TI Sulfation of minoxidil by multiple human cytosolic **sulfotransferases**

AU Anderson, Robert J.; Kudlacek, Patrick E.; Clemens, Dahn L.

CS Sect. Endocrinol. Diabetes Metabolism, Veterans Affairs Med. Cent., Omaha, NE, 68105, USA

SO Chem.-Biol. Interact. (1998), 109(1-3), 53-67
CODEN: CBINA8; ISSN: 0009-2797

PB Elsevier Science Ireland Ltd.

DT Journal

LA English

CC 1 (Pharmacology)

AB Minoxidil is an antihypertensive agent and **hair growth** promoter that is metabolized by sulfation to the active compd., minoxidil sulfate. Thermostable phenol **sulfotransferase** (TS PST or P-PST) was initially thought to catalyze the reaction, and the enzyme was designated minoxidil **sulfotransferase** (MNX-ST). Information about human ST activities toward minoxidil would be useful in developing the

capacity to predict individual responses to minoxidil based on tissue levels of STs. Therefore, human STs was studied from platelet homogenates, partially purified platelets, scalp skin high speed supernatants and COS-1 cell cDNA expressed prepns. using a radiochem. enzymic assay with minoxidil as the substrate. Studies showed the presence of TS PST, TL (thermolabile) PST and MNX-ST activities in human scalp skin. Biochem. properties and correlation studies suggested that in addn. to TS PST, the TL PST activity, another ST activity or both were involved in the reaction. Partially purified human platelet TL PST tested with minoxidil and dopamine showed identical thermal stabilities and similar responses to the inhibitors 2,6-dichloro-4-nitrophenol (DCNP) and NaCl. To characterize the activity of TL PST toward minoxidil, several biochem. properties of the enzyme expressed from a human liver cDNA clone was investigated. When assayed with minoxidil and dopamine, thermal stabilities of the expressed enzyme were identical and IC50 values for the inhibitors DCNP and NaCl were similar. It was also demonstrated that cDNA encoded human liver dehydroepiandrosterone sulfotrans-ferase and estrogen **sulfotransferase** contributed to the sulfation of minoxidil. The results confirm that at least four human STs contribute to minoxidil sulfation. MNX-ST activity represents a combination of ST activities. The data indicate that multiple ST activities should be taken into account in attempts to predict the regulation of minoxidil sulfation and individual responses to minoxidil.

L130 ANSWER 3 OF 97 HCAPLUS COPYRIGHT 1998 ACS DUPLICATE 2
AN 1997:145282 HCAPLUS
DN 126:148537
TI Transdermal and oral treatment of **androgenic** alopecia
IN Crandall, Wilson T.
PA Crandall, Wilson, T., USA
SO PCT Int. Appl., 22 pp.
CODEN: PIXXD2
PI WO 9702041 A1 970123
DS W: AU, BR, CA, JP, MX
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE
AI WO 96-US11270 960703
PRAI US 95-842 950703
US 95-5643 951019
US 96-676095 960702
DT Patent
LA English
IC ICM A61K035-78
ICS A61K039-385; A61K031-35; A61K031-205; A61K031-12
CC 63-6 (**Pharmaceuticals**)
AB This invention relates to the topical and oral treatment of **hair** loss, esp. **androgenic** alopecia, by providing formulations that include anti-**androgens**, esp. exts. of the saw palmetto plant, coenzyme Q, and acetyl carnitine, and optionally simulators of adenylate cyclase to **stimulate hair growth**, to increase the luster of **hair**, and to decrease **hair** graying.
ST **androgenic** alopecia drug compn
IT Alopecia
(**androgenetic**; transdermal and oral compns. for treatment of **androgenic** alopecia)
IT Serenoa repens
(ext. of; transdermal and oral compns. for treatment of **androgenic** alopecia)
IT Essential oils
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(honey almond; transdermal and oral compns. for treatment of

androgenic alopecia)
IT Oral drug delivery systems
Transdermal drug delivery systems
(transdermal and oral compns. for treatment of **androgenic** alopecia)
IT Soya lecithins
Ubiquinones
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(transdermal and oral compns. for treatment of **androgenic** alopecia)
IT 9012-42-4, Adenylate cyclase
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**inhibitors** of; transdermal and oral compns. for treatment of **androgenic** alopecia)
IT 64-17-5, Ethanol, biological studies 111-90-0 142-91-6, Isopropyl palmitate 303-98-0, Coenzyme q10 3079-28-5, n-Decyl methyl sulfoxide 14992-62-2, Acetyl carnitine 24634-61-5, Potassium sorbate 66575-29-9, Forskolin 106392-12-5, Pluronic f127
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(transdermal and oral compns. for treatment of **androgenic** alopecia)

L130 ANSWER 4 OF 97 HCAPLUS COPYRIGHT 1998 ACS
AN 1997:696670 HCAPLUS
DN 128:7304
TI Combination therapy for **androgenic** alopecia with antisense oligonucleotides and minoxidil
IN Hoke, Glenn D. Jr
PA Dyad Pharmaceutical Corporation, USA; Hoke, Glenn D. Jr.
SO PCT Int. Appl., 51 pp.
CODEN: PIXXD2
PI WO 9738728 A1 971023
DS W: AL, AM, AT, AU, AZ, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, US, UZ, VN, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG
AI WO 97-US6133 970414
PRAI US 96-15488 960415
DT Patent
LA English
IC ICM A61K048-00
ICS C07H021-04; C12Q001-68; C12P019-34
CC 63-5 (**Pharmaceuticals**)
Section cross-reference(s): 1
AB Minoxidil has been shown to **stimulate hair growth** or **inhibit** the loss of **hair** in a no. of patients beginning to develop **androgenic** alopecia. The mechanism by which minoxidil (2,4-pyrimidinediamine, 6-(1-piperidinyl)-3-oxide) alters the **hair growth** cycle is uncertain, but is thought to act by increasing vascular circulation to the **hair** follicle. **Inhibitors** of steroid metab., particularly those that **inhibit** the conversion of **testosterone** to **dihydrotestosterone**, have shown effects on hair cycles, including **inhibition** of hair loss. One class of enzymes targeted by these **inhibitors** are the steroid 5-.alpha.-reductases. Minoxidil used in conjunction with effectors of steroid metab., leads to enhanced **hair growth** and decreased rates of **hair** loss. This specification relates to the use of antisense oligonucleotides targeting 5-.alpha.-reductases used in

conjunction with other **hair growth** enhancers and/or **hair** loss **inhibitors.**

ST baldness therapy antisense oligonucleotide minoxidil

IT Creams (drug delivery systems)
Hair follicle
Male pattern baldness
Ointments (drug delivery systems)
Topical drug delivery systems
cDNA sequences
(combination therapy for **androgenic** alopecia with antisense oligonucleotides and minoxidil)

IT Antisense oligonucleotides
RL: BAC (Biological activity or effector, except adverse); PEP (Physical, engineering or chemical process); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(combination therapy for **androgenic** alopecia with antisense oligonucleotides and minoxidil)

IT mRNA
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(steroid 5.alpha.-reductase-specifying; combination therapy for **androgenic** alopecia with antisense oligonucleotides and minoxidil)

IT 38304-91-5, Minoxidil
RL: BAC (Biological activity or effector, except adverse); PEP (Physical, engineering or chemical process); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(combination therapy for **androgenic** alopecia with antisense oligonucleotides and minoxidil)

IT 198718-25-1 198718-26-2 198718-27-3 198718-28-4 198718-29-5
198718-30-8 198718-31-9 198718-32-0 198718-33-1 198718-34-2
198718-35-3 198718-36-4 198718-37-5 198718-38-6 198718-39-7
198718-40-0 198718-41-1 198718-42-2 198718-43-3 198917-50-9
RL: BPR (Biological process); PEP (Physical, engineering or chemical process); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(combination therapy for **androgenic** alopecia with antisense oligonucleotides and minoxidil)

IT 9081-34-9, 5.alpha.-Reductase
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(**inhibition** of; combination therapy for **androgenic** alopecia with antisense oligonucleotides and minoxidil)

L130 ANSWER 5 OF 97 HCAPLUS COPYRIGHT 1998 ACS
AN 1997:759896 HCAPLUS
DN 128:16277
TI **Testosterone** 5.alpha.-reductase **inhibitors** containing Belamcanda chinensis extracts and .alpha.-hydroxy acids and their applications
IN Kawai, Tokuhisa; Hori, Michimasa; Ken, Koh; Ando, Hiroshi
PA Ichimaru Pharcos Inc., Japan
SO Jpn. Kokai Tokkyo Koho, 12 pp.
CODEN: JKXXAF
PI JP 09301884 A2 971125 Heisei
AI JP 96-146823 960515
DT Patent
LA Japanese
IC ICM A61K035-78
ICS A61K007-00; A61K007-06; A61K031-19; C12N009-99; A61K035-78
CC 62-1 (Essential Oils and Cosmetics)
Section cross-reference(s): **63**
AB Skin prepns. for prevention and treatment of acne and **hair**

growth stimulating cosmetics contain title **inhibitors** contg. (A) water, lower alc., or polyol exts. of dried B. chinensis or its rhizome and (B) .alpha.-hydroxy acids or their salts. A lotion was prepd. from sorbitol 2, 1,3-butylene glycol 2, polyethylene glycol 1000 1, polyoxyethylene oleyl ether 2, EtOH 10, 20% EtOH ext. of B. chinensis 10, Na glycolate 0.1, pH adjuster, antiseptic, and H2O to 100 wt.%. Biol. activities of the **inhibitors** were tested.

ST **testosterone** reductase **inhibitor** Belamcanda ext; hydroxy carboxylate **testosterone** reductase **inhibitor**; cosmetic Belamcanda ext hydroxy carboxylate; **hair growth** stimulant Belamcanda hydroxy carboxylate; acne **inhibition** Belamcanda hydroxy carboxylate; alopecia **inhibition** Belamcanda hydroxy carboxylate

IT Lower alcohols
Polyhydric alcohols
RL: NUU (Nonbiological use, unclassified); USES (Uses)
(extn. solvents; **testosterone** reductase **inhibitors** contg. Belamcanda chinensis exts. and .alpha.-hydroxy acids for skin and hair prepns.)

IT Carboxylic acids, biological studies
RL: BAC (Biological activity or effector, except adverse); BUU (Biological use, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(hydroxy; **testosterone** reductase **inhibitors** contg. Belamcanda chinensis exts. and .alpha.-hydroxy acids for skin and hair prepns.)

IT Acne
Alopecia
(**inhibition**; **testosterone** reductase **inhibitors** contg. Belamcanda chinensis exts. and .alpha.-hydroxy acids for skin and hair prepns.)

IT Belamcanda chinensis
Cosmetics
Hair growth stimulants
Topical drug delivery systems
(**testosterone** reductase **inhibitors** contg. Belamcanda chinensis exts. and .alpha.-hydroxy acids for skin and **hair** prepns.)

IT 57-55-6, Propylene glycol, uses 64-17-5, Ethanol, uses 107-88-0, 1,3-Butylene glycol 7732-18-5, Water, uses
RL: NUU (Nonbiological use, unclassified); USES (Uses)
(extn. solvent; **testosterone** reductase **inhibitors** contg. Belamcanda chinensis exts. and .alpha.-hydroxy acids for skin and hair prepns.)

IT 50-21-5, Lactic acid, biological studies 72-17-3, Sodium lactate 77-92-9, Citric acid, biological studies 79-14-1, Glycolic acid, biological studies 676-46-0, Sodium malate 994-36-5, Sodium citrate 2836-32-0, Sodium glycolate 14475-11-7, Sodium tartrate
RL: BAC (Biological activity or effector, except adverse); BUU (Biological use, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**testosterone** reductase **inhibitors** contg. Belamcanda chinensis exts. and .alpha.-hydroxy acids for skin and hair prepns.)

IT 9081-34-9, **Testosterone** 5.alpha.-reductase
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(**testosterone** reductase **inhibitors** contg. Belamcanda chinensis exts. and .alpha.-hydroxy acids for skin and hair prepns.)

L130 ANSWER 6 OF 97 HCAPLUS COPYRIGHT 1998 ACS
KATHLEEN FULLER BT/LIBRARY 308-4290

AN 1997:682194 HCAPLUS
DN 127:336462
TI Lipoxygenase and cyclooxygenase inhibitors for **hair growth** changes preparations
IN Duranton, Albert
PA L'Oreal, Fr.
SO Eur. Pat. Appl., 10 pp.
CODEN: EPXXDW
PI EP 800815 A2 971015
DS R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI
AI EP 97-400727 970328
PRAI FR 96-4795 960417
DT Patent
LA French
IC ICM A61K007-06
CC 62-3 (Essential Oils and Cosmetics)
AB A **hair growth** compn. for the modification of **hair growth** consists of at least 1 lipoxygenase and at least 1 cyclooxygenase inhibitor. Thus, a **hair** lotion contained nordihydroguaiaretic acid 0.10, indomethacin 0.05, propylene glycol 22.80, EtOH 55.10 and water to 100.00 g.
ST **hair growth** lipoxygenase cyclooxygenase inhibitor
IT Carboxylic acids, biological studies
RL: BUU (Biological use, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(aryl; lipoxygenase and cyclooxygenase inhibitors for **hair growth** prepns.)
IT Ginkgo biloba
(exts.; lipoxygenase and cyclooxygenase inhibitors for **hair growth** prepns.)
IT Redox agents
(inhibitor; lipoxygenase and cyclooxygenase inhibitors for **hair growth** prepns.)
IT Eicosanoids
Phenols, biological studies
RL: BUU (Biological use, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(inhibitor; lipoxygenase and cyclooxygenase inhibitors for **hair growth** prepns.)
IT Antioxidants
Hair growth stimulants
Nonsteroidal anti-inflammatory drugs
Shampoos
(lipoxygenase and cyclooxygenase inhibitors for **hair growth** prepns.)
IT Amines, biological studies
Flavonoids
Hydroxy flavones
RL: BUU (Biological use, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(lipoxygenase and cyclooxygenase inhibitors for **hair growth** prepns.)
IT **Hair** preparations
(lotions; lipoxygenase and cyclooxygenase inhibitors for **hair growth** prepns.)
IT 9029-60-1, Lipoxygenase 39391-18-9, Cyclooxygenase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(inhibitor; lipoxygenase and cyclooxygenase inhibitors for **hair growth** prepns.)
IT 50-78-2, Aspirin 52-53-9, Verapamil 53-86-1, Indomethacin 59-67-6D, Nicotinic acid, derivs. 61-68-7, Mefenamic acid 66-71-7, 1,10-Phenanthroline 90-89-1, Diethylcarbamazine

92-43-3, Phenidone 92-84-2D, Phenothiazine, derivs. 94-41-7D, Chalcone, derivs. 95-55-6D, o-Aminophenol, derivs. 120-80-9, Catechol, biological studies 120-80-9D, Catechol, derivs. 121-79-9, Propyl gallate 127-07-1D, derivs. 254-04-6D, Benzopyran, derivs. 288-13-1D, Pyrazole, derivs. 288-32-4D, Imidazole, derivs. 288-47-1D, Thiazole, hydroxy derivs. 327-97-9, Chlorogenic acid 331-39-5, Caffeic acid 394-31-0, 5-Hydroxyanthranilic acid 458-37-7, Curcumin 480-18-2, Dihydroquercetin 480-23-9, Orobol 491-67-8, Baicalein 491-70-3, Luteolin 500-38-9, Nordihydroguaiaretic acid 506-32-1 531-75-9, Esculin **548-83-4**, Galangin 577-85-5, Flavonol 592-88-1, Diallyl sulfide 599-79-1, Sulfasalazine 644-62-2, Meclofenamic acid 745-65-3, PGE1 1321-67-1, Naphthol 5957-80-2, Carnosol 7364-25-2D, Indazolinone, derivs. 7439-89-6D, Iron, chelates 7803-49-8D, Hydroxylamine, derivs. 13345-50-1, PGA2 13745-20-5, 4,2',4'-TrihydroxyChalcone 15307-86-5, Diclofenac 15687-27-1, Ibuprofen 22071-15-4, Ketoprofen 22204-53-1, Naproxen 22494-42-4, Diflunisal 25448-06-0, Octadecatetraenoic acid 26171-23-3, Tolmetine 27686-84-6, Masoprocol 29679-58-1, Fenoprofen 31152-45-1, Eicosatetraenoic acid 32839-18-2, Docosahexaenoic acid 32839-34-2, Docosapentaenoic acid 33922-80-4, Di(1-propenyl) sulfide 36330-85-5, Fenbufen 36441-32-4, 2-Benzyl-1-naphthol 38194-50-2, Sulindac 42924-53-8, Nabumetone 53188-07-1, Trolox C 53716-49-7, Carprofen 56685-04-2, Benzofuranol 59040-30-1, Nafazatrom 59804-37-4, Tenoxicam 60400-92-2, Proxicromil 60940-34-3, Ebselen 65277-42-1, Ketoconazole 65646-68-6 66000-40-6 68012-23-7, Eicosahexaenoic acid 73647-73-1, Viprostol 75207-09-9, Leukotriene C5 79554-19-1 79695-13-9, Leukotriene D5 80445-66-5, Leukotriene B5 84625-61-6, Itraconazole 91431-42-4, Lonapalene 120273-58-7 128484-29-7
RL: BUU (Biological use, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(lipoxygenase and cyclooxygenase inhibitors for **hair growth** prepns.)

L130 ANSWER 7 OF 97 WPIDS COPYRIGHT 1998 DERWENT INFORMATION LTD
AN 97-276677 [25] WPIDS
DNC C97-089115
TI Hair treatment agent - comprises extract of Serenoa repens, saw palmetto.
DC B04 D21
PA (TMCK-N) TMC KAKEN KK
CYC 1
PI JP 09100220 A 970415 (9725)* 3 pp A61K007-06 <--
ADT JP 09100220 A JP 96-116837 960510
PRAI JP 95-112099 950510
IC ICM **A61K007-06**
ICS **A61K007-00; A61K035-78**
AB JP09100220 A UPAB: 970619
Hair treatment agent comprises an extract of Serenoa repens, saw palmetto.
Extract of fruits of Serenoa repens, saw palmetto is preferably used for preparation of the hair treatment agent.
USE - The agent is used for treatment of prostate hypertrophy for **hair growth stimulation** by inactivation of 5-alpha-**reductase** and **inhibition** of 5-alpha-dihydro **testosterone** (5-alpha-DHT).
Dwg.0/0
FS CPI
FA AB
MC CPI: B04-A10; B14-D02; B14-D05D; B14-N07A; B14-R02; D08-B03

L130 ANSWER 8 OF 97 WPIDS COPYRIGHT 1998 DERWENT INFORMATION LTD

AN 97-209235 [19] WPIDS
DNC C97-067317
TI Epithelial cell growth promoter - useful against skin ageing, for skin smoothing and as antiinflammatory and wound healing agent.
DC B02
PA (KIKK) KIKKOMAN CORP; (NODA) ZH NODA SANGYO KAGAKU KENKYUSHO
CYC 1
PI JP 09059166 A 970304 (9719)* 6 pp A61K035-78
ADT JP 09059166 A JP 95-230682 950817
PRAI JP 95-230682 950817
IC ICM A61K035-78
ICS A61K031-70
AB JP09059166 A UPAB: 970512
Epithelial cell growth promoter useful as dermal agent comprises malonyl **isoflavone** glycoside prepd. from soybean or aq. extract of soybean as the active ingredient.
USE - The growth promoter is useful as a skin cosmetic, stimulator of **hair growth**, antiinflammatory agent, for preventing skin ageing, skin smoothing and for wound healing.
Dwg.0/1
FS CPI
FA AB; DCN
MC CPI: B06-A01; B14-C03; B14-L01; B14-N17; B14-R01; B14-R02

L130 ANSWER 9 OF 97 WPIDS COPYRIGHT 1998 DERWENT INFORMATION LTD
AN 98-011640 [02] WPIDS
DNC C98-004167
TI Treatment and prophylaxis of hair loss - especially associated with telogen effluvium, comprises administration of L-lysine.
DC B05
IN RUSHTON, D H
PA (BIOS-N) BIO-SCIENTIFIC LTD; (BIOS-N) BIO-SCI LTD
CYC 76
PI GB 2314019 A 971217 (9802)* 15 pp A61K031-195 <--
WO 9747276 A1 971218 (9805) EN 18 pp A61K007-06 <--
RW: AT BE CH DE DK EA ES FI FR GB GH GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE GH HU IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK TJ TM TR TT UA UG US UZ VN YU
AU 9730404 A 980107 (9820) A61K007-06 <--
ADT GB 2314019 A GB 96-12108 960610; WO 9747276 A1 WO 97-GB1542 970606; AU 9730404 A AU 97-30404 970606
FDT AU 9730404 A Based on WO 9747276
PRAI GB 96-12108 960610
IC ICM **A61K007-06**
ICA A61K031-195
ICI A61K031:505, A61K031:565, A61K031:57, A61K031:5
AB GB 2314019 A UPAB: 980112
Use of L-lysine (I) for the prophylaxis and treatment of hair loss is new provided that (I) is not in the form of a complex with a transition metal and that (I) is not used together with a combination of trigonelline and vitamin B6, a combination of divalent iron, pantothenic acid and methionine, and/or garlic oil or garlic extract. Preferably, (I) is the sole active agent. Also claimed is a kit comprising containers of active agents useful for treating genetic hair loss, including (I) together with minoxidil, anti-**androgens** (II), 5 alpha -**reductase inhibitors**, aromatase **inhibitors** and/or corticosteroids.
USE - The method is particularly useful for treating telogen effluvium (claimed) and suboptimal **hair growth**.

(I) also improves the efficacy of known treatments for genetic hair loss, including anagen-dependent alopecia, **androgenic** alopecia, **androgenetic** alopecia, common baldness, female baldness, diffuse hair loss and male pattern baldness. Dosage of (I) is 200-2000 (preferably 500-1500) mg/day, administered orally in 1-3 doses.

ADVANTAGE - Treatment with (I) results in a substantial **increase** in **hair growth** and a **reduction** in the amount of hair shed.

Dwg.0/0

FS CPI
FA AB; DCN
MC CPI: B01-C03; B01-C04; B01-C05; B07-D05; B07-D12; B10-B01B; B14-D02A; B14-D05D; B14-D07A; B14-R02

L130 ANSWER 10 OF 97 BIOSIS COPYRIGHT 1998 BIOSIS
AN 97:513456 BIOSIS
DN 99812659
TI Central precocious puberty and chronic renal failure: A reversible condition post renal transplantation.
AU Loh K-C; Salisbury S R; Accott P; Gillis R; Crocker J F
CS Dep. General Med., Tan Tock Seng Hospital, Moulmein Road, Singapore 308433, Singapore
SO Journal of Pediatric Endocrinology & Metabolism 10 (5). 1997. 539-545.
LA English
PR Biological Abstracts Vol. 104 Iss. 012 Ref. 170263
AB A 3 year-old boy with chronic renal failure associated with prune belly syndrome who developed central precocious puberty is described. He had been maintained on cyclic peritoneal dialysis from age 13 months with creatinine levels of 400-600 mu-mol/l. Increased linear **growth** rate probably began at 18 months, and by 38 months of age he had testicular enlargement and pubic **hair** consistent with Tanner stage 2. Elevated levels of serum **testosterone** (3.6 nmol/l; normal lt 0.7 nmol/l) and luteinizing hormone (LH) (2.8 IU/l; normal lt 1.0 IU/1) were demonstrated with a pubertal response to luteinizing hormone-releasing hormone (LHRH) **stimulation** (peak LH 43.5 IU/l). Other endocrine tests demonstrated hyperprolactinemia (170 mu-g/l; normal 3.4-22 mu-g/l), but normal pituitary-thyroid and pituitary-adrenal functions and normal cranial MR imaging. Despite LHRH-agonist therapy with leuprolide over the next 8 months, he showed an incomplete response with only partial **inhibition** of basal LH and **testosterone** levels, and continued significant increments in height standard deviation scores (Ht-SDS) and bone age estimates. However, the sexual precocity appeared fully reversible following a successful living-related renal transplant at age 50 months. Despite discontinuation of leuprolide treatment post-operatively, there was a full reversal of his serum LH and **testosterone** to a prepubertal profile as well as normalization of the serum prolactin levels. Whereas most boys with chronic renal failure show delayed pubertal development and suppressed linear **growth**, our patient presents a unique phenomenon of reversible central precocious puberty. The effects of leuprolide therapy in the presence of a uremic milieu and the outcome of successful renal transplantation on sexual precocity are described.
ST RESEARCH ARTICLE; HUMAN; PRESCHOOL; MALE; PATIENT; CENTRAL PRECOCIOUS PUBERTY; CHRONIC RENAL FAILURE; PRUNE BELLY SYNDROME; **TESTOSTERONE**; LUTEINIZING HORMONE; LHRH; HYPERPROLACTINEMIA; LEUPROLIDE; LHRH AGONIST-DRUG; BONE AGE; HEIGHT; RENAL TRANSPLANTATION; PROLACTIN; CLINICAL ENDOCRINOLOGY; NEPHROLOGY; PEDIATRICS; ENDOCRINE DISEASE-GONADS; UROLOGIC DISEASE; CONGENITAL DISEASE; MUSCLE DISEASE; METABOLIC DISEASE; TRANSPLANTATION METHOD; THERAPEUTIC METHOD; SURGICAL METHOD

RN 58-22-0 (TESTOSTERONE)
9002-62-4 (PROLACTIN)
9002-67-9 (LUTEINIZING HORMONE)
9034-40-6 (LHRH)
53714-56-0 (LEUPROLIDE)
CC Anatomy and Histology, General and Comparative-Regeneration and Transplantation *11107
Urinary System and External Secretions-General; Methods *15501
Endocrine System-General *17002
Pharmacology-General *22002
Pediatrics *25000
BC Hominidae 86215

L130 ANSWER 11 OF 97 HCAPLUS COPYRIGHT 1998 ACS
AN 1997:20097 HCAPLUS
DN 126:114963
TI Characterization of recombinant human liver dehydroepiandrosterone **sulfotransferase** with minoxidil as the substrate
AU Kudlacek, Patrick E.; Clemens, Dahn L.; Halgard, Christine M.; Anderson, Robert J.
CS SECTION OF ENDOCRINOLOGY, DIABETES AND METABOLISM, DEPARTMENT OF VETERANS AFFAIRS MEDICAL CENTER, OMAHA, NE, USA
SO Biochem. Pharmacol. (1997), 53(2), 215-221
CODEN: BCPCA6; ISSN: 0006-2952
PB Elsevier
DT Journal
LA English
CC 7-3 (Enzymes)
AB Biotransformation of xenobiotics and hormones through sulfate conjugation is an important metabolic pathway in humans. The activation of minoxidil, an antihypertensive agent and **hair growth** stimulator, by sulfation (sulfonation) is carried out by more than one **sulfotransferase**. Initially only the thermostable form of phenol **sulfotransferase** was thought to catalyze minoxidil sulfation. We document in this report the new finding that human liver dehydroepiandrosterone **sulfotransferase** (DHEA ST), an hydroxysteroid **sulfotransferase** distinct from phenol **sulfotransferases**, also catalyzes the reaction. To characterize more precisely the activity of DHEA ST toward minoxidil, we used COS-1 cells to express DHEA ST from a human liver cDNA clone. The apparent Km values for minoxidil and [35S]3'-phosphoadenosine-5'-phosphosulfate were 3.9 mM and 0.13 .mu.M, resp. The 50% inactivation temp. of the COS-expressed enzyme was 42.degree., and the IC50 value for 2,6-dichloro-4-nitrophenol was 1.4 .times. 10-4 M. Both the thermal stability behavior and response to DCNP were similar when the cDNA encoded DHEA ST was assayed with DHEA or minoxidil as a substrate. NaCl led to a greater activation of the cDNA-expressed DHEA ST when assayed with DHEA (2.5-fold) than when the same prepn. was assayed with minoxidil (1.4-fold). These data indicate that DHEA ST catalyzes the sulfate conjugation of minoxidil. DHEA ST activity present in the human gut and liver would be expected to add to the overall sulfate conjugation of orally administered minoxidil. Thus, DHEA ST activity must be considered when detg. the human tissue **sulfotransferase** contribution to minoxidil sulfation.
ST liver dehydroepiandrosterone **sulfotransferase** minoxidil
IT Enzyme kinetics
Liver
(sulfation of minoxidil by recombinant human liver dehydroepiandrosterone **sulfotransferase**)
IT 9032-76-2, Dehydroepiandrosterone **sulfotransferase**
RL: BAC (Biological activity or effector, except adverse); BIOL (Biological study)

(sulfation of minoxidil by recombinant human liver dehydroepiandrosterone **sulfotransferase**)

IT 38304-91-5, Minoxidil
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(sulfation of minoxidil by recombinant human liver dehydroepiandrosterone **sulfotransferase**)

L130 ANSWER 12 OF 97 HCAPLUS COPYRIGHT 1998 ACS DUPLICATE 3
AN 1997:60829 HCAPLUS
DN 126:54751
TI A Comparison of Phenobarbital and Codeine Incorporation into Pigmented and Nonpigmented Rat **Hair**
AU Gygi, Steven P.; Wilkins, Diana G.; Rollins, Douglas E.
CS Center for Human Toxicology Department of Pharmacology and Toxicology, University of Utah, Salt Lake City, UT, 84112, USA
SO J. Pharm. Sci. (1997), 86(2), 209-214
CODEN: JPMSAE; ISSN: 0022-3549
PB American Chemical Society
DT Journal
LA English
CC 1-11 (Pharmacology)
OS CJACS-IMAGE; CJACS
AB Drugs and endogenous compds. circulating in the blood may ultimately become incorporated into a **growing hair** shaft. **Hair** anal. for drugs of abuse is a **growing** field in the area of forensic and clin. toxicol. However, the underlying principles that govern drug incorporation into **hair** are not known. In this study, we examd. the incorporation of a weak acid, phenobarbital, and a weak base, codeine, into Sprague-Dawley (SD) rat **hair**. Codeine or phenobarbital was administered to male SD rats at 40 mg/kg/day for 5 days by i.p. (i.p.) injection. **Hair** was collected from the back 14 days after beginning the 5-day dosing protocol and analyzed by gas chromatog./mass spectrometry (GC/MS) for codeine and phenobarbital. The time-courses of phenobarbital and codeine in plasma were also obtained after a single i.p. injection (40 mg/kg). Concns. of codeine and phenobarbital in SD **hair** samples were 0.98 .+-. 0.10 and 17.01 .+-. 1.40 ng/mg **hair**, resp. The areas under the curve (AUC) of plasma concn. vs. time for codeine and phenobarbital were 1.58 and 414.50 .mu.g h/.mu.L, resp. Notwithstanding the greater phenobarbital concns. in **hair**, when plasma concns. were considered, codeine was apparently incorporated to a 15-fold greater extent than phenobarbital. Because **hair** pigmentation may be important in drug incorporation, the incorporation of these two drugs was also studied in Long-Evans (LE; produces both black and white **hair** on the same animal) rats after 40 mg/kg/day of i.p. drug administration for 5 days. **Hair** was collected at the same time as the previous expt. Concns. of codeine in **hair** were 44-times greater in pigmented than nonpigmented **hair** from the same animals. In contrast, **hair** concns. of phenobarbital were identical in both pigmented and nonpigmented **hair**. These data suggest that **hair** pigmentation greatly affects weak base incorporation but not weak acid incorporation into **hair**. Because **hair** concns. of phenobarbital are not affected by pigmentation, phenobarbital may be an ideal drug to sep. out factors other than pigmentation involved in incorporation of drugs into **hair**.
ST phenobarbital codeine sedative **hair**
IT Hypnotics and Sedatives
(comparison of phenobarbital and codeine incorporation into pigmented and non-pigmented rat **hair**)
IT **50-06-6**, Phenobarbital, biological studies 76-57-3,

Codeine
RL: ADV (Adverse effect, including toxicity); BAC (Biological activity or effector, except adverse); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(comparison of phenobarbital and codeine incorporation into pigmented and non-pigmented rat **hair**)

L130 ANSWER 13 OF 97 HCAPLUS COPYRIGHT 1998 ACS
AN 1997:594411 HCAPLUS
DN 127:273210
TI Specific inhibition of **hair** follicle formation by epidermal **growth** factor in an organ culture of developing mouse skin
AU Kashiwagi, Mariko; Kuroki, Toshio; Huh, Nam-Ho
CS Department of Cancer Cell Research, Institute of Medical Science, University of Tokyo, Shirokanedai, 108, Japan
SO Dev. Biol. (1997), 189(1), 22-32
CODEN: DEBIAO; ISSN: 0012-1606
PB Academic
DT Journal
LA English
CC 2-10 (Mammalian Hormones)
AB Embryonic mouse skin undergoes a drastic morphol. change from 13 to 16 gestational days, i.e., formation of rudiments of **hair** follicles and stratification and cornification of interfollicular epidermis. To investigate underlying mol. mechanisms of the morphogenesis, the authors established an organ culture system that allows skin tissues isolated from 12.5- or 13.5-days postcoitus embryos to develop in a manner that is histol. and temporally similar to the process in vivo. Expression of differentiation markers of epidermal keratinocytes including cholesterol **sulfotransferase** and cytokeratin K1 was induced in culture, as it occurs also in vivo. The morphogenic process was obsd. by time-lapse videomicrog. In this culture system, epidermal **growth** factor (EGF) and transforming **growth** factor .alpha. specifically and completely inhibited the **hair** follicle formation with marginal effects on interfollicular epidermis. The inhibitory action by EGF was reversible and stage specific, i.e., at an early stage of the development of **hair** rudiments. Among known ligands to the EGF receptor, Schwannoma-derived **growth** factor and heparin-binding EGF were expressed in in vivo epidermis during the period of the initial formation of **hair** follicles. EGF receptor is expressed in epidermis throughout the developing period examd. Using an adenovirus vector, the authors demonstrated that the lacZ gene was transduced into the epidermal and dermal cell layers without appreciable toxicity. These results indicate that the present culture system provides a unique opportunity to investigate mol. mechanisms of skin morphogenesis including the role of EGF signaling under defined exptl. conditions.
ST EGF **hair** follicle skin morphogenesis culture
IT Cell differentiation
Embryogenesis (animal)
Epidermis (skin)
Hair follicle
Keratinocyte
Morphogenesis (animal)
Skin
Tissue culture (animal)
(EGF specific inhibition of **hair** follicle formation in developing mouse skin in culture)
IT Transforming **growth** factor .alpha.
RL: BAC (Biological activity or effector, except adverse); BIOL (Biological study)

(EGF specific inhibition of **hair** follicle formation in developing mouse skin in culture)

IT Epidermal **growth** factor receptors
RL: BOC (Biological occurrence); BPR (Biological process); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(EGF specific inhibition of **hair** follicle formation in developing mouse skin in culture)

IT Keratins
RL: BPR (Biological process); BUU (Biological use, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative); PROC (Process); USES (Uses)
(keratin 1; EGF specific inhibition of **hair** follicle formation in developing mouse skin in culture)

IT **Growth** factors (animal)
RL: BOC (Biological occurrence); BIOL (Biological study); OCCU (Occurrence)
(schwannoma-derived **growth** factors; EGF specific inhibition of **hair** follicle formation in developing mouse skin in culture)

IT 62229-50-9, Epidermal **growth** factor
RL: BAC (Biological activity or effector, except adverse); BIOL (Biological study)
(EGF specific inhibition of **hair** follicle formation in developing mouse skin in culture)

IT 154531-34-7
RL: BOC (Biological occurrence); BIOL (Biological study); OCCU (Occurrence)
(EGF specific inhibition of **hair** follicle formation in developing mouse skin in culture)

IT 9032-76-2, Cholesterol **sulfotransferase**
RL: BPR (Biological process); BUU (Biological use, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative); PROC (Process); USES (Uses)
(EGF specific inhibition of **hair** follicle formation in developing mouse skin in culture)

L130 ANSWER 14 OF 97 BIOSIS COPYRIGHT 1998 BIOSIS
AN 97:131247 BIOSIS
DN 99423060
TI History of **hair** analysis.
AU Sachs H
CS Inst. Legal Med., Univ. Munich, Frauenlobstr. 7a, 80337 Munich, Germany
SO Forensic Science International 84 (1-3). 1997. 7-16. ISSN: 0379-0738
LA English
PR Biological Abstracts Vol. 103 Iss. 007 Ref. 095203
ST HISTORICAL ARTICLE; HUMAN; GUINEA-PIG; FORENSICS; TOXICOLOGY; FORENSIC TOXICOLOGY; **HAIR**; HISTORY; RADIOIMMUNOASSAY; MORPHINE; COCAINE; METABOLISM; AMPHETAMINE; THIN LAYER CHROMATOGRAPHY; CODEINE; URINE; PHENOBARBITAL; BLOOD; INTEGUMENTARY SYSTEM; GC-MS; GAS CHROMATOGRAPHY-MASS SPECTROMETRY; HEROIN; METHAMPHETAMINE; METABOLITE; BENZOYLECGONINE; ECGONINE; METHYLECGONINE; NORCOCAINE; COCAETHYLENE; NORCOCAETHYLENE; THC; TETRAHYDROCANNABINOL; CARBOXY-TETRAHYDROCANNABINOL; DRUG ABUSE; INTEGUMENTARY SYSTEM; ANALYTICAL METHOD; FLUORESCENCE DETECTION; EXCRETORY SYSTEM; BLOOD AND LYMPHATICS
RN **50-06-6** (PHENOBARBITAL)
50-36-2 (COCAINE)
57-27-2 (MORPHINE)
76-57-3 (CODEINE)
300-62-9 (AMPHETAMINE)
481-37-8 (ECGONINE)
519-09-5 (BENZOYLECGONINE)
529-38-4 (COCAETHYLENE)

537-46-2 (METHAMPHETAMINE)
561-27-3 (HEROIN)
1972-08-3 (TETRAHYDROCANNABINOL)
7143-09-1 (METHYLECGONINE)
18717-72-1 (NORCOCAINE)
137220-02-1 (NORCOCAETHYLENE)
CC General Biology-History and Archaeology *00522
General Biology-Forensic Science *00531
Radiation-General *06502
Behavioral Biology-Human Behavior *07004
Biochemical Methods-General *10050
Biochemical Studies-General *10060
Biophysics-General Biophysical Studies *10502
Metabolism-General Metabolism; Metabolic Pathways *13002
Blood, Blood-Forming Organs and Body Fluids-General; Methods *15001
Urinary System and External Secretions-General; Methods *15501
Integumentary System-General; Methods *18501
Psychiatry-Addiction-Alcohol, Drugs, Smoking, etc. *21004
Pharmacology-General *22002
Toxicology-General; Methods and Experimental *22501
Immunology and Immunochemistry-General; Methods *34502
BC **Hominidae 86215**
Caviidae 86300

L130 ANSWER 15 OF 97 HCAPLUS COPYRIGHT 1998 ACS DUPLICATE 4
AN 1996:660913 HCAPLUS
DN 125:293042
TI Use of angiogenesis suppressors for inhibiting **hair growth**
IN Ahluwalia, Gurpreet S.; Styczynski, Peter; Shander, Douglas
PA Handelman, Joseph H., USA
SO PCT Int. Appl., 23 pp.
CODEN: PIXXD2
PI WO 9626712 A2 960906
DS W: AL, AM, AT, AU, AZ, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI
RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, DE, DK, ES, FR, GA, GB, GR, IE, IT, LU, MC, ML, NL, PT, SE
AI WO 96-US2790 960227
PRAI US 95-396446 950228
DT Patent
LA English
IC ICM A61K007-48
CC 1-12 (Pharmacology)
Section cross-reference(s): 62
AB A method of inhibiting **hair growth** in a mammal includes applying, to an area of skin from which reduced **hair growth** is desired, a dermatol. acceptable compn. contg. a non-steroidal suppressor of angiogenesis. The effective compds. include **sulfotransferase** inhibitors, heparin binding antagonists, Cu chelators, histidine decarboxylase inhibitors, mast cell degranulation inhibitors, histamine receptor antagonists, ACE inhibitors, angiotensin II receptor antagonists, prostaglandin synthetase inhibitors, NK1 receptor antagonists, PAF receptor antagonists, and cytochrome P 450 reductase inhibitors. A topical prepn. contg. 10 % bathocuproine, was applied to male intact Golden Syrian hamsters; **hair growth** was inhibited by 81 %.
ST angiogenesis suppressor **hair growth** inhibition; **hirsutism** angiogenesis inhibitor; topical prepn bathocuproine **hair growth** inhibition
IT **Hair** preparations

Hirsutism
(angiogenesis suppressors for inhibiting **hair growth**)

IT Protamines
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(angiogenesis suppressors for inhibiting **hair growth**)

IT Mast cell
(degranulation inhibitors; angiogenesis suppressors for inhibiting **hair growth**)

IT Blood vessel
(formation of; angiogenesis suppressors for inhibiting **hair growth**)

IT Proteoglycans, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(heparin-contg., antagonists; angiogenesis suppressors for inhibiting **hair growth**)

IT Pentosans
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(sulfates, angiogenesis suppressors for inhibiting **hair growth**)

IT Kinin receptors
Receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(tachykinin NK1, antagonists; angiogenesis suppressors for inhibiting **hair growth**)

IT Glycoproteins, specific or class
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(thrombospondins, angiogenesis suppressors for inhibiting **hair growth**)

IT Pharmaceutical dosage forms
(topical, angiogenesis suppressors for inhibiting **hair growth**)

IT 67-43-6, Diethylenetriamine pentaacetic acid 83-89-6, Quinacrine 91-81-6, Tripelennamine 113-92-8 120-80-9, 1,2-Benzenediol, biological studies 1398-62-5, Chitin sulfate 1845-11-0, Nafoxidine 3316-09-4, p-Nitrocatechol 4431-00-9, Aurintricarboxylic acid 4733-39-5, Bathocuproine 7491-74-9, Piracetam 10540-29-1, Tamoxifen 12772-57-5, Radicicol 15826-37-6, Cromoglycate 18550-55-5, Hyponitric acid 21829-25-4, Nifedipine 23110-15-8, Fumagillin 23593-75-1, Clotrimazole 24280-93-1, Mycophenolic acid 25614-03-3, Bromocryptine 37270-94-3, Platelet factor-4 38096-31-0D, Diaminoanthraquinone, derivs. 50679-08-8, Terfenadine 51481-61-9, Cimetidine 52698-84-7, Bathocuproinesulfonate 57381-26-7, Irsogladine **65899-73-2**, Tioconazole 70050-43-0, .alpha.-Fluoromethylhistidine 75847-73-3, Enalapril 76547-98-3, Lisinopril 84088-42-6, Linomide 110590-61-9 114798-26-4, Losartan 126509-46-4, Eponemycin 129912-34-1 135911-02-3 182930-58-1
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(angiogenesis suppressors for inhibiting **hair growth**)

IT 51-45-6, Histamine, biological studies 11128-99-7, Angiotensin II 33507-63-0, Substance P 65154-06-5, Platelet activating factor
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(antagonists; angiogenesis suppressors for inhibiting **hair growth**)

IT 9015-82-1, Angiotensin-converting enzyme 9023-09-0, **Sulfotransferase** 9024-61-7, Histidine decarboxylase

9039-06-9, Cytochrome P450 reductase 9055-65-6, Prostaglandin synthetase
RL: BSU (Biological study, unclassified); BIOL (Biological study) (inhibitors; angiogenesis suppressors for inhibiting **hair growth**)

L130 ANSWER 16 OF 97 HCAPLUS COPYRIGHT 1998 ACS
AN 1997:113419 HCAPLUS
DN 126:122303
TI **Hair growth** promoting compositions containing isoflavanoid derivatives
IN Kung, Patrick C.; Li, Ze Zeng
PA Kung, Patrick, C., USA
SO PCT Int. Appl., 20 pp.
CODEN: PIXXD2
PI WO 9639832 A1 961219
DS W: AL, AM, AU, AZ, BB, BG, BR, BY, CA, CN, CZ, EE, FI, GE, HU, IL, IS, JP, KG, KP, KR, KZ, LK, LR, LS, LT, LV, MD, MG, MK, MN, MX, NO, NZ, PL, RO, RU, SG, SI, SK, TJ, TM, TR, TT, UA, UZ, VN, AM, AZ, BY
RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG
AI WO 96-US8433 960603
PRAI US 95-484097 950607
US 96-659466 960531
DT Patent
LA English
IC ICM A01N043-16
ICS A61K031-35
CC 62-3 (Essential Oils and Cosmetics)
Section cross-reference(s): 1, 26, 63
OS MARPAT 126:122303
AB Novel compns. of isoflavanoid derivs. useful for the treatment of male pattern baldness and **alopecia** areata, promoting the conversion of gray **hair** to the original pigment in **hair** follicles, and increasing the blood supply to the brain are disclosed. The invention also relates to methods for treatment of male pattern baldness and **alopecia** areata, gray **hair**, and brain circulatory deficiencies. Sodium methoxide 6.48 was added to 50 mL DMF and the mixt. was distd. to eliminate alc. then, resulting product was cooled to .ltoreq.20.degree.. Dimethylamino-methoxy sulfuric acid Me ester (prepn. given) was added dropwise to the cooled product and the mixt. was allowed to react for 5 h. The reaction mixt. was distd. to remove dimethyllformaide from the mixt. followed by addn. of water to obtain daidzein (I). A tablet contained I 100, lactose 50, starch 23, microcryst. cellulose 2, dicalcium phosphate 30 mg, surfactants trace, and magnesium trace. The efficacy of tablets (2 tablet 3 times/day) in treatment of hypertensive male bald subject is reported.
ST **hair growth** promotor isoflavanoid deriv; pharmaceutical tablet daidzein male baldness
IT **Alopecia**
(areata; **hair growth** promoting compns. contg. isoflavanoid derivs.)
IT Cerebrovascular diseases
Creams (drug delivery systems)
Male pattern baldness
Ointments (drug delivery systems)
Tablets (drug delivery systems)
(**hair growth** promoting compns. contg. isoflavanoid derivs.)
IT **Hair growth** stimulants
RL: BUU (Biological use, unclassified); SPN (Synthetic preparation);

THU (Therapeutic use); BIOL (Biological study); PREP (Preparation);
USES (Uses)
(**hair growth** promoting compns. contg.
isoflavanoid derivs.)
IT Isoflavonoids
RL: RCT (Reactant)
(**hair growth** promoting compns. contg.
isoflavanoid derivs.)
IT **485-72-3P** 486-63-5P **486-66-8P**, Daidzein
19725-36-1P 56401-04-8P 89019-85-2P 139256-06-7P
142574-14-9P 146307-82-6P 148356-24-5P 186246-60-6P
186246-61-7P 186246-62-8P 186246-63-9P 186246-64-0P
186246-65-1P 186246-66-2P 186246-67-3P 186246-68-4P
186246-69-5P
RL: BUU (Biological use, unclassified); SPN (Synthetic preparation);
THU (Therapeutic use); BIOL (Biological study); PREP (Preparation);
USES (Uses)
(**hair growth** promoting compns. contg.
isoflavanoid derivs.)
IT 68-12-2, reactions 75-93-4, Methyl sulfate 186246-70-8
RL: RCT (Reactant)
(**hair growth** promoting compns. contg.
isoflavanoid derivs.)

L130 ANSWER 17 OF 97 HCAPLUS COPYRIGHT 1998 ACS
AN 1996:449455 HCAPLUS
DN 125:95532
TI method and apparatus for **hair growth** promotion
IN Okamura, Katsumasa
PA Mohatsu Kurinitsukuriibu Nijui, Japan
SO Jpn. Kokai Tokkyo Koho, 9 pp.
CODEN: JKXXAF
PI JP 08107936 A2 960430 Heisei
AI JP 94-246376 941012
DT Patent
LA Japanese
IC ICM A61N001-40
ICS A61K007-06
CC 62-3 (Essential Oils and Cosmetics)
Section cross-reference(s): 1
AB An app. for **hair growth** promotion comprises a high frequency comb-contg., high frequency-based ozone-generating device and a low frequency comb-contg., low frequency-based **stimulating** device. A method for **hair growth** promotion involves: application of a herbal medicine-based **hair growth** stimulant to the scalp, simulation with th low frequency-based **stimulating** device to promote penetration of the **hair growth** stimulants into the **hair** root, and treatment with the high frequency device to activate the cells or tissues located between the epidermal and dermal layers and to irradiate the scalp with ozone to **inhibit** male alopecia-related 5.alpha.-**dehydrotestosterone** formation.
ST app **hair growth** promotion ozone
IT Ozonizers
(in app. for **hair growth** promotion with **hair growth** stimulants and ozone)
IT Alopecia
(male; method and app. for **hair growth** promotion with **hair growth** stimulants and ozone)
IT Apparatus
(method and app. for **hair growth** promotion with **hair growth** stimulants and ozone)

IT **Pharmaceutical** natural products
RL: BUU (Biological use, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(method and app. for **hair growth** promotion with herbal medicine-based **hair growth** stimulants and ozone)
IT **Hair** preparations
(**growth** stimulants, method and app. for **hair growth** promotion with herbal medicine-based **hair growth** stimulants and ozone)
IT Plant
(medicinal, method and app. for **hair growth** promotion with herbal medicine-based **hair growth** stimulants and ozone)
IT 521-18-6, 5.alpha.-**Dihydrotestosterone**
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(**inhibition** of; in method and app. for **hair growth** promotion with **hair growth** stimulants and ozone)
IT 10028-15-6, Ozone, biological studies
RL: BUU (Biological use, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(method and app. for **hair growth** promotion with herbal medicine-based **hair growth** stimulants and ozone)

L130 ANSWER 18 OF 97 HCAPLUS COPYRIGHT 1998 ACS
AN 1996:209989 HCAPLUS
DN 124:241803
TI Skin-conditioning compositions containing isoflavone
IN Brunke, Reinhold A.
PA New Standard Gmbh, Germany
SO Ger. Offen., 4 pp.
CODEN: GWXXBX
PI DE 4432947 A1 960321
AI DE 94-4432947 940916
DT Patent
LA German
IC ICM A61K007-48
ICS A61K007-06; A61K031-35
ICI A61K031-35, A61K031-56; A61K031-70, A61K031-56
CC 62-4 (Essential Oils and Cosmetics)
AB Skin care compns. contg. isoflavone and its derivs. act as radical scavengers which prevent aging of the skin, as dermal angiogenesis inhibitors, and as antiproliferative agents against melanomas, and are useful for treatment of varicose veins, acne, fatty skin, graying of the **hair**, pigment spots, and **alopecia**. Thus, a gel for treatment of acne was prepd. by combining a mixt. of Eumulgin B1 3, Cetiol 868 10, methylparaben 0.15, propylparaben 0.10, and soybean ext. (source of isoflavones) 10.0 wt.% with H2O 73, Sepigel 305 3.5, and Kathon CG 0.05 wt.%.
ST skin conditioner isoflavone; acne treatment isoflavone; angiogenesis skin isoflavone; baldness treatment isoflavone
IT Blood vessel
(formation of dermal, inhibitors; skin-conditioning compns. contg. isoflavones)
IT Soybean
(isoflavones of; skin-conditioning compns. contg. isoflavones)
IT Radicals, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(scavengers; skin-conditioning compns. contg. isoflavones)
IT Seborrhea
(skin-conditioning compns. contg. isoflavones)

IT Acne
Alopecia
(treatment of; skin-conditioning compns. contg. isoflavones)
IT Cosmetics
(conditioners, skin-conditioning compns. contg. isoflavones)
IT Skin, disease
(couperose, treatment of; skin-conditioning compns. contg. isoflavones)
IT **Hair** preparations
(**growth** stimulants, skin-conditioning compns. contg. isoflavones)
IT Skin, disease
(hyperpigmentation, macular, treatment of; skin-conditioning compns. contg. isoflavones)
IT Flavonoids
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(iso-, oxo, skin-conditioning compns. contg. isoflavones)
IT Neoplasm inhibitors
(melanoma, skin-conditioning compns. contg. isoflavones)
IT Skin, disease
(oily, treatment of; skin-conditioning compns. contg. isoflavones)
IT Skin, disease
(spider, vascular, treatment of; skin-conditioning compns. contg. isoflavones)
IT 446-72-0, 5,7,4'-Trihydroxyisoflavone 480-23-9, 3',4',5,7-Tetrahydroxyisoflavone **486-66-8**, 7,4'-Dihydroxyisoflavone 491-80-5, 5,7-Dihydroxy-4'-methoxyisoflavone 529-59-9, Genistin 529-60-2 548-76-5 552-66-9, Daidzin 574-12-9, Isoflavone 574-12-9D, Isoflavone, derivs. 2284-31-3 34086-51-6
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(skin-conditioning compns. contg. isoflavones)

L130 ANSWER 19 OF 97 WPIDS COPYRIGHT 1998 DERWENT INFORMATION LTD
AN 96-425097 [42] WPIDS
DNC C96-133886
TI **Reduction** of mammalian **hair growth** -
by topical admin. of a compsn. contg. a catechin cpd..
DC B02 D21
IN AHLUWALIA, G S
PA (HAND-I) HANDELMAN J H; (AHLU-I) AHLUWALIA G S
CYC 72
PI WO 9626705 A1 960906 (9642)* EN 18 pp A61K007-06 <--
RW: AT BE CH DE DK EA ES FR GB GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG
W: AL AM AT AU AZ BB BG BR BY CA CH CN CZ DE DK EE ES FI GB GE HU IS JP KE KG KP KR KZ LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK TJ TM TR TT UA UG US UZ VN
AU 9651781 A 960918 (9701) A61K007-06 <--
ZA 9601599 A 961129 (9702) 19 pp A61K000-00 <--
US 5674477 A 971007 (9746) 4 pp A61K007-06 <--
EP 814754 A1 980107 (9806) EN A61K007-06 <--
R: AT BE CH DE DK ES FR GB GR IE IT LI LU NL PT SE
ADT WO 9626705 A1 WO 96-US2791 960227; AU 9651781 A AU 96-51781 960227; ZA 9601599 A ZA 96-1599 960228; US 5674477 A US 95-396426 950228; EP 814754 A1 EP 96-908589 960227, WO 96-US2791 960227
FDT AU 9651781 A Based on WO 9626705; EP 814754 A1 Based on WO 9626705
PRAI US 95-396426 950228
REP 2.Jnl.Ref ; FR 2527927; FR 2708851; JP 2202581; JP 62053917; WO 9324106
IC ICM **A61K000-00; A61K007-06**

AB WO 9626705 A UPAB: 961021
The following are claimed: (A) **Reducing** mammalian **hair growth**, comprising: (a) selecting an area of skin from which **reduced hair growth** is desired; and (b) applying a compsn. including a catechin cpd. to the area. (B) **Reducing** mammalian **hair growth**, comprising: (a) selecting an area of skin from which **reduced hair growth** is desired; and (b) applying a compsn. comprising green tea leaves (or a component extracted from green tea leaves) to the area.
USE - The process is esp. useful for **reducing androgen-stimulated hair growth** (e.g. as in female hirsutism).
ADVANTAGE - The catechin cpds. do not cause side effects, and the process also avoids problems associated with shaving or plucking, such as cutting or skin irritation.
Dwg.0/0
FS CPI
FA AB; DCN
MC CPI: B04-A09A; B04-A10B; B06-A01; B14-R01; D08-B03; D08-B09A

L130 ANSWER 20 OF 97 WPIDS COPYRIGHT 1998 DERWENT INFORMATION LTD
AN 96-209168 [21] WPIDS
DNC C96-066667
TI **Hair growth** stimulating or loss inhibiting agents - comprising e.g. copper salt, **flavone** cpd., xanthine cpd., muco-polysaccharide, vitamin and/or plant extract..
DC A96 B05 D21
IN BARTON, S P; GALLEY, E
PA (BOOT) BOOTS CO PLC
CYC 65
PI WO 9610387 A2 960411 (9621)* EN 15 pp A61K007-06
RW: AT BE CH DE DK ES FR GB GR IE IT KE LU MC MW NL OA PT SD SE SZ UG
W: AM AT AU BB BG BR BY CA CH CN CZ DE DK EE ES FI GB GE HU IS JP KE KG KP KR KZ LK LR LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK TJ TM TT UA UG US UZ VN
AU 9537441 A 960426 (9631) A61K007-06
WO 9610387 A3 960613 (9633) A61K007-06
ADT WO 9610387 A2 WO 95-EP3861 950928; AU 9537441 A AU 95-37441 950928; WO 9610387 A3 WO 95-EP3861 950928
FDT AU 9537441 A Based on WO 9610387
PRAI GB 94-19715 940930
REP No-SR.Pub ; 1.Jnl.Ref ; DE 2901452; DE 3724259; DE 4225985; EP 250300; EP 334486; FR 1476532; FR 2282856; FR 2310767; FR 2587208; GB 2106386; GB 807787; JP 07010720; WO 8202833; WO 9415574
IC ICM A61K007-06
ICS A61K035-78
AB WO 9610387 A UPAB: 960529
Use of one or more of the following as hair stimulants is new: (a) a **flavone** or deriv., suitably comprising rutin, (e.g. troxerutin); (b) a water-soluble potassium, copper and/or zinc salt, suitably an acetate; (c) a xanthine (e.g. a theophylline) or a deriv. (e.g. methyl silanol theophylline acetate alginate (MSTAA)); (d) a mucopolysaccharide or deriv. (e.g. dimethylsilanol hyaluronate (DMSH)); (e) a fat-soluble vitamin or deriv. (e.g. vitamin A palmitate or vitamin E); (f) zedoary, ginger and/or cinnamon oil; and (g) an allyl-based plant extract (e.g. onion or garlic extract), e.g. onion extract in coconut oil or garlic extract in butylene glycol.
USE - (a)-(g) are useful for inhibiting hair loss (e.g. alopecia areata) and/or stimulating **hair growth** in humans, esp. on the scalp, and are useful for medical and/or cosmetic purposes.

I have document

Dwg.0/0
FS CPI
FA AB; DCN
MC CPI: A12-V01; A12-V04A; B03-A; B04-A06; B04-A10F; B04-B01C1; B04-C02; B05-A03A; B06-A01; B14-R02; D08-B03

L130 ANSWER 21 OF 97 WPIDS COPYRIGHT 1998 DERWENT INFORMATION LTD
AN 96-200693 [20] WPIDS
DNC C96-063364
TI **Inhibiting hair growth** with protein kinase C **inhibitor** - applied topically, partic. for control of female hirsutism.
DC B05 D21
IN AHLUWALIA, G S; SHANDER, D; STYCZYNSKI, P
PA (HAND-I) HANDELMAN J H; (AHLU-I) AHLUWALIA G S; (SHAN-I) SHANDER D; (STYC-I) STYCZYNSKI P
CYC 67
PI WO 9609806 A2 960404 (9620)* EN 14 pp A61K007-06 <--
RW: AT BE CH DE DK ES FR GB GR IE IT KE LU MC MW NL OA PT SD SE SZ UG
W: AM AT AU BB BG BR BY CA CH CN CZ DE DK EE ES FI GB GE HU IS JP KE KG KP KR KZ LK LR LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK TJ TM TT UA UG US UZ VN
AU 9537230 A 960419 (9630) A61K007-06 <--
ZA 9508145 A 960626 (9631) 14 pp A61K000-00 <--
US 5554608 A 960910 (9642) 5 pp A61K031-55 <--
EP 783292 A1 970716 (9733) EN A61K007-06 <--
R: AT BE CH DE DK ES FR GB GR IE IT LI LU MC NL PT SE
ADT WO 9609806 A2 WO 95-US12134 950921; AU 9537230 A AU 95-37230 950921; ZA 9508145 A ZA 95-8145 950927; US 5554608 A US 94-314327 940928; EP 783292 A1 EP 95-935068 950921, WO 95-US12134 950921
FDT AU 9537230 A Based on WO 9609806; EP 783292 A1 Based on WO 9609806
PRAI US 94-314327 940928
REP No-SR.Pub
IC ICM **A61K000-00; A61K007-06; A61K031-55**
ICS **A61K031-47; A61K031-505; A61K031-54**
AB WO 9609806 A UPAB: 960520
Inhibition of **hair growth** in mammals comprises applying to the appropriate area of skin, a compsn. contg. a protein kinase C (PKC) **inhibitor** (I).
USE - The compsn. is partic. used to **reduce growth** of facial **hair** in women with hirsutism or similar conditions, esp. where growth is **stimulated** by **androgens**.
Dwg.0/0
FS CPI
FA AB; DCN
MC CPI: B04-M01; B14-R02; D08-B07

L130 ANSWER 22 OF 97 WPIDS COPYRIGHT 1998 DERWENT INFORMATION LTD
AN 96-179695 [18] WPIDS
DNC C96-056651
TI Use of aromatase **inhibitor, androgen** receptor antagonist or pre-oestrogen as cosmetic agent - to maintain or **increase hair growth** or to **reduce hair growth**, e.g. in treatment of hirsutism or as depilatory agents, also method to detect whether patient will benefit from treatment.
DC B04 B05 D16 D21
IN MESSENGER, A G
PA (UYSH-N) UNIV SHEFFIELD; (UYSH-N) UNIV CENT SHEFFIELD HOSPITALS NHS TRUST
CYC 66
PI WO 9608231 A1 960321 (9618)* EN 39 pp A61K007-06 <--

RW: AT BE CH DE DK ES FR GB GR IE IT KE LU MC MW NL OA PT SD SE SZ UG
W: AM AT AU BB BG BR BY CA CH CN CZ DE DK EE ES FI GB GE HU IS JP KE KG KP KR KZ LK LR LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK TJ TM TT UA UG US UZ VN
GB 2295088 A 960522 (9624) 37 pp A61K007-06 <--
AU 9535253 A 960329 (9628) A61K007-06 <--
EP 777458 A1 970611 (9728) EN A61K007-06 <--
R: AT BE CH DE DK ES FR GB GR IE IT LI LU MC NL PT SE

ADT WO 9608231 A1 WO 95-GB2166 950913; GB 2295088 A GB 95-18725 950913; AU 9535253 A AU 95-35253 950913; EP 777458 A1 EP 95-932057 950913, WO 95-GB2166 950913

FDT AU 9535253 A Based on WO 9608231; EP 777458 A1 Based on WO 9608231

PRAI GB 94-18547 940915; GB 94-18484 940914

REP 6.Jnl.Ref ; DE 2840144; DE 3615396; DE 3621757; EP 163490; EP 566979; JP 61018711; JP 62103005; US 4684635; WO 8502543; WO 8601402; WO 8602269

IC ICM **A61K007-06**
ICS C07K016-40

AB WO 9608231 A UPAB: 960503
The following are claimed, e.g.:(a) the use of an aromatase **inhibitor** (AI) as a cosmetic agent;(b) a method for treating or preventing hair loss comprising administering an AI to an area to be treated;(c) an antibody for use in preventing hair loss, raised against an AI, and(d) the use of an AI in the mfr. of a prepn. for the **redn.** in the regression of **hair growth** or in the alleviation of baldness.
USE - The AI and ARA compsns. can be used to induce, maintain or **increase hair growth** and reverse, arrest or prevent the onset of baldness. Pre-oestrogen compsns. can be used to **increase** oestrogen concns. and **reduce hair growth**, e.g. in the treatment of hirsutism or as depilatory agents (all claimed).
Dwg.0/0

FS CPI

FA AB; DCN

MC CPI: B04-F02; B04-G01; B04-L08; B11-C08E1; B12-K04A; B14-D01B; B14-D02; B14-D10; B14-R02; D05-A02; D05-H09; D08-B03; D08-B07

L130 ANSWER 23 OF 97 BIOSIS COPYRIGHT 1998 BIOSIS DUPLICATE 5

AN 97:162197 BIOSIS

DN 99461400

TI Examination of stability of anticonvulsants in a protease solution and assay of anticonvulsants in hairs.

AU Fujii J; Higashi A; Nakano M

CS Dep. Pharmaceutical Services, Kumamoto Univ. Hosp., 1-1-1 Honjo, Kumamoto 860, Japan

SO Biological & Pharmaceutical Bulletin 19 (12). 1996. 1614-1617. ISSN: 0918-6158

LA English

PR Biological Abstracts Vol. 103 Iss. 008 Ref. 117069

AB For analyzing the concentrations of drugs in hairs, a new method of digestion of hairs with Biopurase, a protease obtained from Bacillus subtilis, was examined. The concentrations of drugs in hairs were then determined in order to examine the usefulness of the protease for the digestion of hairs. The stability of five anticonvulsants in the protease solution was maintained over a 12-h period. In the clinical tests, the concentrations of the drugs in hairs obtained from patients who were taking anticonvulsants for a long time were determined. The concentration of phenobarbital in hairs in 10 patients taking phenobarbital ranged from 194 to 5020 ng/10 mg with a mean of 578 ng/10 mg, and the concentration of phenytoin in hairs in 6 patients taking phenytoin ranged from 44 to 299 ng/10 mg with a mean of 115 ng/10 mg. From these results, the transfer of

phenobarbital and phenytoin from circulating blood into hairs was confirmed, and the usefulness of Biopurase for the digestion of hairs was proved.
ST RESEARCH ARTICLE; BACILLUS SUBTILIS; HUMAN; **HAIR**; **HAIR** ANALYSIS; PHENOBARBITAL; ANTICONVULSANT-DRUG; PHENYTOIN; ANTICONVULSANT-DRUG; BIOPURASE; BACTERIAL PROTEASE; PHARMACOLOGY; METHODOLOGY; DRUG CONCENTRATION; INTEGUMENTARY SYSTEM; ANALYTICAL METHOD
RN **50-06-6** (PHENOBARBITAL)
57-41-0 (PHENYTOIN)
9001-92-7 (PROTEASE)
CC Biochemical Methods-General *10050
Biochemical Studies-General *10060
Integumentary System-General; Methods *18501
Pharmacology-General *22002
BC Endospore-forming Gram-Positives 07810
Hominidae 86215

L130 ANSWER 24 OF 97 HCAPLUS COPYRIGHT 1998 ACS
AN 1995:668443 HCAPLUS
DN 123:122729
TI **Hair growth** stimulants containing flavanonols
IN Oochi, Atsushi; Wakayama, Micho; Kidena, Hidefumi; Hirayama, Noriko; Hotsuta, Mitsuyuki; Imokawa, Genji; Kanazawa, Satoshi; Nishizawa, Yoshinori; Ichinose, Susumu
PA Kao Corp, Japan
SO Jpn. Kokai Tokkyo Koho, 7 pp.
CODEN: JKXXAF
PI JP 07112916 A2 950502 Heisei
AI JP 93-278631 931108
PRAI JP 93-213203 930827
DT Patent
LA Japanese
IC ICM A61K007-06
CC 62-3 (Essential Oils and Cosmetics)
Section cross-reference(s): 1, 63
AB **Hair growth** stimulants contain flavanonol, its derivs., and/or their glycosides as active ingredients. **Hair** follicle tissues of rats were cultured in the presence of 10 ng/mL taxifolin to show 119% DNA-forming activity, vs. 100%, for controls. Formulation examples are given.
ST **hair growth** stimulant flavanonol; glycoside flavanonol **hair growth** stimulant
IT Dandruff
(control of; **hair growth** stimulants contg. flavanonols (glycosides))
IT Glycosides
RL: BAC (Biological activity or effector, except adverse); BUU (Biological use, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(flavanonol; **hair growth** stimulants contg. flavanonols (glycosides))
IT Inflammation inhibitors
Vasodilators
(**hair growth** stimulants contg. flavanonols (glycosides) and vasodilators or inflammation inhibitors)
IT **Alopecia**
(treatment of; **hair growth** stimulants contg. flavanonols (glycosides))
IT **Hair** preparations
(antidandruff, **hair growth** stimulants contg. flavanonols (glycosides))
IT **Hair** preparations
(**growth** stimulants, **hair growth**

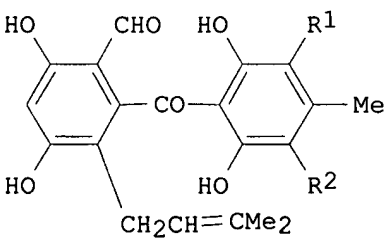
stimulants contg. flavanonols (glycosides))

IT Flavonoids
RL: BAC (Biological activity or effector, except adverse); BUU (Biological use, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(oxo dihydro hydroxy, **hair growth** stimulants contg. flavanonols (glycosides))

IT 480-13-7, Alpinone 480-18-2, Taxifolin 480-20-6, Aromadendrin 490-31-3 492-00-2, 7-Hydroxyflavonol 520-18-3 548-82-3 **548-83-4** 572-31-6, Engeletin 1226-22-8, Garbanzol 4382-33-6, Dihydrorobinetin 4382-36-9 6068-78-6, 3',4'-Dihydroxyflavonol 14919-49-4, 4'-Hydroxyflavonol 18422-83-8, Dihydromorin 20725-03-5, Fustin 27200-12-0, Ampeloptin 29838-67-3, Astilbin 30987-58-7, Isoengeletin 34198-87-3 37971-69-0 37971-70-3 55568-97-3, trans-3-Hydroxyflavanone 166376-01-8
RL: BAC (Biological activity or effector, except adverse); BUU (Biological use, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**hair growth** stimulants contg. flavanonols (glycosides))

IT 1406-18-4, Vitamin E 23327-65-3 52225-20-4, DL-.alpha.-Tocopherol acetate
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(**hair growth** stimulants contg. flavanonols (glycosides) and vasodilators or inflammation inhibitors)

L130 ANSWER 25 OF 97 HCAPLUS COPYRIGHT 1998 ACS
AN 1995:584215 HCAPLUS
DN 123:8034
TI 2-(3-Methyl-2-butenyl)benzophenones, their fungal manufacture, and **testosterone**-5.alpha.-reductase **inhibitors**, **hair growth** stimulants, and UV absorbers containing them
IN Wachi, Yoji; Yamashita, Toyonobu; Komatsu, Kazuo; Yoshida, Seiichi
PA Shiseido Co Ltd, Japan
SO Jpn. Kokai Tokkyo Koho, 14 pp.
CODEN: JKXXAF
PI JP 07061950 A2 950307 Heisei
AI JP 93-207818 930823
DT Patent
LA Japanese
IC ICM C07C049-86
ICS A61K007-06; A61K007-42; A61K031-12; C12N009-99; C12P007-24
ICI C12P007-24, C12R001-645
CC 16-2 (Fermentation and Bioindustrial Chemistry)
Section cross-reference(s): 62, **63**
OS MARPAT 123:8034
GI



I

AB The title compds.(I, R1-2 = H, halo) are manufd. by culture of Chrysosporium spp. (filamentous fungi). **Testosterone** -5.alpha.-reductase (II) **inhibitors, hair growth** stimulants, and UV absorbers contg. I are also claimed. Chrysosporium sp. 87G2 (FERM P-1370) was cultured in a medium contg. glucose, potato starch, Asn, and salts under agitation at 30.degree. for 5 days to give I (R1 = R2 = Cl) (III). IC50 value of III on II was 10 .mu.M. EtOH 60.0, III 0.5, propylene glycol 2.0 wt.%, perfume, perfume solubilizer, and H2O balance were mixed to give a **hair growth** stimulant.

ST benzophenone deriv **testosterone** reductase **inhibitor**; Chrysosporium benzophenone manuf **hair grower; hair growth** stimulant
benzophenone deriv; UV absorbent benzophenone deriv fermn; methylbutenylbenzophenone fermn **testosterone** reductase **inhibitor**

IT Shampoos
(**hair growth-stimulating**; manuf. of (methylbutenyl)benzophenones as **testosterone** reductase **inhibitors** with Chrysosporium and their uses as **hair growth** stimulants and UV absorbers)

IT Fermentation
Sunscreens
(manuf. of (methylbutenyl)benzophenones as **testosterone** reductase **inhibitors** with Chrysosporium and their uses as **hair growth** stimulants and UV absorbers)

IT Chrysosporium
(strain 87G2 (FERM P-13705); manuf. of (methylbutenyl)benzophenones as **testosterone** reductase **inhibitors** with Chrysosporium and their uses as **hair growth** stimulants and UV absorbers)

IT **Hair** preparations
(**growth** stimulants, manuf. of (methylbutenyl)benzophenones as **testosterone** reductase **inhibitors** with Chrysosporium and their uses as **hair growth** stimulants and UV absorbers)

IT 9081-34-9, **Testosterone**-5.alpha.-reductase
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(**inhibitors**; manuf. of (methylbutenyl)benzophenones as **testosterone** reductase **inhibitors** with Chrysosporium and their uses as **hair growth** stimulants and UV absorbers)

IT 163768-82-9P 163768-83-0P
RL: BAC (Biological activity or effector, except adverse); BMF (Bioindustrial manufacture); BPR (Biological process); BUU (Biological use, unclassified); BIOL (Biological study); PREP (Preparation); PROC (Process); USES (Uses)
(manuf. of (methylbutenyl)benzophenones as **testosterone** reductase **inhibitors** with Chrysosporium and their uses as **hair growth** stimulants and UV absorbers)

L130 ANSWER 26 OF 97 HCAPLUS COPYRIGHT 1998 ACS
AN 1995:557432 HCAPLUS
DN 122:299059
TI **Hair growth** stimulants comprising lipoxygenase or cycloxygenase stimulants or inhibitors
IN Duranton, Albert; De Lacharriere, Olivier
PA Oreal S. A., Fr.
SO Eur. Pat. Appl., 13 pp.
CODEN: EPXXDW
PI EP 648488 A1 950419
DS R: DE, ES, FR, GB, IT
AI EP 94-402055 940914

PRAI FR 93-12178 931013
DT Patent
LA French
IC ICM A61K031-00
ICS A61K031-05; A61K031-495; A61K031-095; A61K031-12; A61K031-405; A61K035-78; A61K007-06
CC 63-3 (Pharmaceuticals)
AB The title compns. contg. lipoxygenase or cycloxygenase stimulants or inhibitors are disclosed. A **hair** lotion contained nordihydroguaiaretic acid 0.1, linoleic acid 0.1, propylene glycol 22.8, EtOH 95.degree. 55.1, and water q.s. 100g.
ST **hair growth** stimulant lipoxygenase stimulant inhibitor; cycloxygenase stimulant inhibitor **hair growth** stimulant; lotion nordihydroguaiaretic acid **hair growth** stimulant
IT Leukotrienes
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(C5, B5, and D5; **hair growth** stimulants comprising lipoxygenase or cycloxygenase stimulants or inhibitors)
IT Terpenes and Terpenoids, biological studies
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(biol. studies; boswellic acids, **hair growth** stimulants comprising lipoxygenase or cycloxygenase stimulants or inhibitors)
IT Ginkgo biloba
(exts.; **hair growth** stimulants comprising lipoxygenase or cycloxygenase stimulants or inhibitors)
IT Antioxidants
Chelating agents
Shampoos
(**hair growth** stimulants comprising lipoxygenase or cycloxygenase stimulants or inhibitors)
IT Anthocyanins
Flavanols
Flavonoids
Hydroxamic acids
Lymphokines and Cytokines
Phosphatidylethanolamines
Phosphatidylglycerols
Phosphatidylinositols
Phosphatidylserines
Phenols, biological studies
Phosphatidylcholines, biological studies
Phospholipids, biological studies
Sulfides, biological studies
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(**hair growth** stimulants comprising lipoxygenase or cycloxygenase stimulants or inhibitors)
IT Eicosanoids
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(inhibitors; **hair growth** stimulants comprising lipoxygenase or cycloxygenase stimulants or inhibitors)
IT Prostaglandins
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(A, **hair growth** stimulants comprising lipoxygenase or cycloxygenase stimulants or inhibitors)
IT Fatty acids, biological studies

RL: BUU (Biological use, unclassified); BIOL (Biological study);
USES (Uses)
(C>20-polyunsatd., **hair growth** stimulants
comprising lipoxygenase or cycloxygenase stimulants or
inhibitors)

IT Prostaglandins
RL: BUU (Biological use, unclassified); BIOL (Biological study);
USES (Uses)
(E, **hair growth** stimulants comprising
lipoxygenase or cycloxygenase stimulants or inhibitors)

IT Carboxylic acids, biological studies
RL: BUU (Biological use, unclassified); BIOL (Biological study);
USES (Uses)
(aryl, esters, **hair growth** stimulants
comprising lipoxygenase or cycloxygenase stimulants or
inhibitors)

IT Ion channel
(calcium, interfering agents; **hair growth**
stimulants comprising lipoxygenase or cycloxygenase stimulants or
inhibitors)

IT **Hair** preparations
(**growth** stimulants, **hair growth**
stimulants comprising lipoxygenase or cycloxygenase stimulants or
inhibitors)

IT Cosmetics
(lotions, **hair growth** stimulants comprising
lipoxygenase or cycloxygenase stimulants or inhibitors)

IT Peptides, biological studies
RL: BUU (Biological use, unclassified); BIOL (Biological study);
USES (Uses)
(neuropeptides, **hair growth** stimulants
comprising lipoxygenase or cycloxygenase stimulants or
inhibitors)

IT Inflammation inhibitors
(nonsteroidal, **hair growth** stimulants
comprising lipoxygenase or cycloxygenase stimulants or
inhibitors)

IT Animal **growth** regulators
RL: BUU (Biological use, unclassified); BIOL (Biological study);
USES (Uses)
(.beta.-transforming **growth** factors, **hair**
growth stimulants comprising lipoxygenase or
cycloxygenase stimulants or inhibitors)

IT 62031-54-3, Fibroblast **growth** factor
RL: BUU (Biological use, unclassified); BIOL (Biological study);
USES (Uses)
(beta; **hair growth** stimulants comprising
lipoxygenase or cycloxygenase stimulants or inhibitors)

IT 52-53-9, Verapamil 53-86-1, Indomethacin 59-67-6D, Nicotinic
acid, derivs. 60-33-3, Linoleic acid, biological studies
70-18-8, Glutathion, biological studies 90-89-1,
Diethylcarbamazine 92-43-3, Phenidone 92-84-2D, Phenothiazine,
derivs. 94-41-7D, Chalcone, derivs. 95-55-6 121-79-9,
Propylgallate 127-07-1 254-04-6D, Benzopyran, derivs.
288-13-1D, Pyrazole, derivs. 327-97-9, Chlorogenic acid
331-39-5, Caffeic acid 394-31-0, 5-Hydroxyanthranilic acid
458-37-7, Curcumin 463-40-1, .alpha.-Linolenic acid 480-18-2,
Dihydroquercetin 480-23-9, Orobol 491-67-8, Baicalein
491-70-3, Luteolin 500-38-9, Nordihydroguaiaretic acid 506-32-1
531-75-9, Esculin **548-83-4**, Galangin 592-88-1, Diallyl
sulfide 599-79-1, Sulfasalazine 1321-67-1, Naphthol 1783-84-2,
Dihomo-.gamma.-linolenic acid 5957-80-2, Carnosol 6039-97-0D,
2(3H)-Thiazolone, derivs. 6581-66-4D, derivs. 6590-43-8
7364-25-2, Indazolinone 7803-49-8, Hydroxylamine, biological

studies 7803-49-8D, Hydroxylamine, alkyl derivs. 10102-43-9, Nitrogen oxide (NO), biological studies 10418-03-8, Stanozolol 12678-01-2, Phenanthroline 14542-13-3D, alkyl derivs. 27686-84-6, Masoprocol 31152-45-1, Eicosatetraenoic acid 32839-18-2, Docosahexaenoic acid 32839-30-8, Eicosapentaenoic acid 32839-34-2, Docosapentaenoic acid 33922-80-4, Di(1-propenyl) sulfide 36441-32-4, 2-Benzyl-1-naphthol 56685-04-2, Benzofuranol 59040-30-1, Nafazatrom 60400-92-2, Proxicromil 62229-50-9, Epidermal **growth** factor 65154-06-5, Platelet activating factor 65277-42-1, Ketoconazole 65646-68-6 66000-40-6 73180-00-4, 15-Hydroxyeicosatetraenoic acid 73647-73-1, Viprostol 74237-20-0, 6-Chloro-2,3-dihydroxy-1,4-naphthoquinone 81275-46-9, Octa-decatetraenoic acid 82451-61-4 84625-61-6, Itraconazole 91431-42-4, Lonapalene 111406-87-2, Zileuton 120273-58-7 163121-02-6D, derivs.
RL: BUU (Biological use, unclassified); BIOL (Biological study);
USES (Uses)
(**hair growth** stimulants comprising lipoxygenase or cycloxygenase stimulants or inhibitors)

IT 506-32-1D, Arachidonic acid, derivs.
RL: BUU (Biological use, unclassified); BIOL (Biological study);
USES (Uses)
(metabolites; **hair growth** stimulants comprising lipoxygenase or cycloxygenase stimulants or inhibitors)

IT 39391-18-9, Cyclooxygenase 63551-74-6, Lipoxygenase
RL: BUU (Biological use, unclassified); BIOL (Biological study);
USES (Uses)
(stimulants or inhibitors; **hair growth** stimulants comprising lipoxygenase or cycloxygenase stimulants or inhibitors)

L130 ANSWER 27 OF 97 WPIDS COPYRIGHT 1998 DERWENT INFORMATION LTD
AN 95-328081 [42] WPIDS
DNC C95-145519
TI **Inhibiting hair growth** in mammals - using ornithine amino transferase **inhibitor**, esp. for cosmetic **inhibition** of facial hair.
DC B05 D16 E14 E16
IN THOMPSON, L W; WALLACE, H M; WISLER, M M; WU, J; FUNKHOUSER, M G; SHANDER, D
PA (BAKO) BAKER HUGHES INC; (HAND-I) HANDELMAN J H; (FUNK-I) FUNKHOUSER M G; (SHAN-I) SHANDER D
CYC 65
PI WO 9524181 A1 950914 (9542)* EN 15 pp A61K007-06 <--
RW: AT BE CH DE DK ES FR GB GR IE IT KE LU MC MW NL OA PT SD SE SZ UG
W: AM AT AU BB BG BR BY CA CH CN CZ DE DK EE ES FI GB GE HU JP KE KG KP KR KZ LK LR LT LU LV MD MG MN MW MX NL NO NZ PL PT RO RU SD SE SG SI SK TJ TM TT UA UG US UZ VN
AU 9519816 A 950925 (9601) G01V003-30
AU 9521172 A 950925 (9601) A61K007-06 <--
US 5474763 A 951212 (9604) 3 pp A61K007-06 <--
ZA 9502031 A 960228 (9614) 13 pp A61K000-00 <--
EP 754024 A1 970122 (9709) EN A61K007-06 <--
R: AT BE CH DE DK ES FR GB GR IE IT LI LU NL PT SE
JP 09510210 W 971014 (9751) 13 pp A61K007-06 <--
MX 9603923 A1 970401 (9821) A61K007-06 <--
ADT WO 9524181 A1 WO 95-US2915 950308; AU 9519816 A AU 95-19816 950307; AU 9521172 A AU 95-21172 950308; US 5474763 A US 94-212012 940311; ZA 9502031 A ZA 95-2031 950310; EP 754024 A1 EP 95-913991 950308, WO 95-US2915 950308; JP 09510210 W JP 95-523629 950308, WO 95-US2915 950308; MX 9603923 A1 MX 96-3923 960906
FDT AU 9519816 A Based on WO 9524663; AU 9521172 A Based on WO 9524181;

EP 754024 A1 Based on WO 9524181; JP 09510210 W Based on WO 9524181
PRAI US 94-212012 940311; US 94-212194 940311; US 94-212257 940314;
US 94-212269 940314; US 94-214343 940314; US 94-214916 940314
REP WO 8602269; WO 9421216; WO 9421217
IC ICM **A61K000-00; A61K007-06**
ICS **A61K007-15; A61K007-155; A61K031-19**
AB WO 9524181 A UPAB: 951026
Mammalian **hair growth** is **inhibited** by applying to a selected area of the skin a compsn. contg. an **inhibitor** (I) of ornithine aminotransferase (OAT).
Also new are compsns. contg. (I) and a dermatological vehicle or carrier. Compsns. are partic. used in cosmetics to **inhibit hair growth** on the face. (I) partic. **inhibit androgen stimulates hair growth**, e.g. in cases of female hirsutism.
(I) is pref. 6-fluoro-2,5-diamino hexanoic acid; (S)-2-amino-4-amino oxy-butyric acid or 3-amino-2,3-dihydro benzoic acid (which are irreversible **inhibitors**).
These contain 1-30% (I) plus a spreadable vehicle or carrier. (I) is applied at 100-3000 mug/cm2 of skin, typically once or twice a day for at least 3 months. The treatment causes a **redn.** in growth of at least 30 (best at least 70)% in the Golden Syrian hamster assay.
Dwg.0/0
FS CPI
FA AB; DCN
MC CPI: B04-C03B; B10-A11B; B10-A18; B10-B01B; B10-B02E; B10-E04B; B10-E04C; B12-M02F; B14-D02; B14-D06; B14-N17; D05-C03; D08-B03; E10-B01C; E10-B02A

L130 ANSWER 28 OF 97 WPIDS COPYRIGHT 1998 DERWENT INFORMATION LTD
AN 95-350220 [45] WPIDS
CR 93-167280 [20]
DNC C95-153479
TI **Reducing** rate of mammalian **hair growth** - by applying organic **inhibitor** of L-asparagine synthetase, used for treating hirsutism, etc..
DC B05
IN AHLUWALIA, G S
PA (AHLU-I) AHLUWALIA G S
CYC 1
PI US 5444090 A 950822 (9545)* 3 pp A61K031-225 <--
ADT US 5444090 A CIP of US 91-788168 911105, US 94-212584 940311
PRAI US 94-212584 940311; US 91-788168 911105
IC ICM **A61K031-225**
ICS **A61K031-19; A61K031-195**
AB US 5444090 A UPAB: 951114
Reducing the rate of mammalian **hair growth** comprises applying to an area of skin a compsn. contg. organic **inhibitor** of L-asparagine synthetase.
The compsn. pref. contains a dermatologically acceptable vehicle in which the concn. of the **inhibitor** is 1-30 wt.%. The **inhibitor** is guanidino succinic acid, oxaloacetic acid, cysteine sulphinic acid, diethylaminomalonate or ethacrynic acid. The **inhibitor** is a reversible or an irreversible **inhibitor**. When the compsn. is tested in the Golden Syrian hamster assay, the **redn.** in **hair growth** is 23.3%, esp. 52.6%.
USE - The method is esp. useful in **reducing** the rate of human **hair growth**, e.g. on the leg, arm, armpit, torso or face, esp. the beard. It may be used on women suffering from hirsutism. It may be used to **reduce androgen-stimulated hair growth** . The amt. of **inhibitor** applied to the skin is 100-3,000

mug/cm2.
Dwg.0/0
FS CPI
FA AB; DCN
MC CPI: B10-A09C; B10-A17; B10-B02J; B10-C02; B10-C03; B10-C04B; B10-D03; B14-D10; B14-R02

L130 ANSWER 29 OF 97 WPIDS COPYRIGHT 1998 DERWENT INFORMATION LTD
AN 96-021386 [03] WPIDS
DNC C96-007419
TI Medicated shampoo for hair care and treatment - contg. lipoic acid or deriv. and synergist(s), e.g. selenium salt or vitamin..
DC B05 D21
IN SCHINDLER, H; ULRICH, H; WEISCHER, C H
PA (ASTA) ASTA MEDICA AG
CYC 1
PI DE 4419783 A1 951207 (9603)* 6 pp A61K007-06 <--
ADT DE 4419783 A1 DE 94-4419783 940606
PRAI DE 94-4419783 940606
IC ICM **A61K007-06**
AB DE 4419783 A UPAB: 960122
A shampoo (I) for treatment and care of hair contains at least the following active agents: (A) oxidised or **reduced** enantiomers of alpha-lipoic acid, dihydrolipoic acid (racemate) or their esters, 6,8-bis-nor-lipoic acid, tetra-nor-lipoic acid, or 1,2-dithiacyclopentane-3-butylsulphonic acid or their alkali metal salts, at a concn. of 0.2-10%; and (B) one or more combination partners such as selenium salts, disodium salts, potassium salts of a condensation product of lauric acid and protein hydrolysate, palm-kernel fatty acid sarcoside of methyltaurine, palm kernel oil fatty acid sarcoside of triethanolamine, sodium salt of a condensation product of undecylenic acid, water-soluble vitamin E or F, ascorbic acid, beer extract, camomile flower extract or dye concentrates.
USE - (I) is useful for treating **hair** loss, **hair growth** disorders, cytostatic-induced alopecia, **hair** brittleness, dandruff with dry or oily seborrhoea, impetignous eczema and pyodermia of the scalp, serborrhoeic eczema of the hair base and seborrhoeic associated symptoms of **androgenetic** alopecia, and for **increasing** the lifetime of hair (all claimed).
ADVANTAGE - (A) and (B) have a synergistic therapeutic effect, esp. in protection of elastin (a component of the connective tissue of the scalp). alpha-lipoic acid also **inhibits** catabolic enzymes, due to its antiphlogistic and calcium scavenging activity.
Dwg.0/0
FS CPI
FA AB; DCN
MC CPI: B03-F; B03-H; B05-A01A; B05-A01B; B05-B02C; B07-B03; B10-C04E; B14-R02; D08-B03; D08-B04

L130 ANSWER 30 OF 97 BIOSIS COPYRIGHT 1998 BIOSIS
AN 96:39407 BIOSIS
DN 98611542
TI Comparison of a gonadotropin-releasing hormone agonist and a low dose oral contraceptive given alone or together in the **treatment** of hirsutism.
AU Heiner J S; Greendale G A; Kawakami A K; Lapolt P S; Fisher M; Young D; Judd H L
CS Dep. Obstetics Gynecol., Olive View-University California-Los Angeles Med. Cent., 14445 Olive View Drive, 2B163 Sylmar, CA 91342, USA
SO Journal of Clinical Endocrinology & Metabolism 80 (12). 1995. 3412-3418. ISSN: 0021-972X
LA English

PR Biological Abstracts Vol. 101 Iss. 003 Ref. 039247
AB Chronic GnRH agonist **therapy** lowers **androgens** and decreases **androgen**-dependent **hair** shaft diameter, but the resulting induction of hypoestrogenemia has limited its usefulness as a single agent. Estrogen- and progestin-containing oral contraceptives also **reduce** circulating **androgen** levels and are commonly used empirically for the **treatment** of hirsutism, but have not been evaluated in a blinded randomized controlled fashion. The present study is the first double masked trial to evaluate the combination use of a GnRH agonist and an estrogen-containing oral contraceptive and tests our hypothesis that these could synergistically **reduce androgen** levels and suppress hormone-dependent **hair growth** while avoiding the symptoms and risks of agonist-induced hypoestrogenemia. We enrolled 64 women in a 24-week blinded randomized controlled trial to compare placebo, nafarelin (NAF; 400 mu-g, intranasal spray, twice daily), norethindrone (1 mg), and ethinyl estradiol (NOR 1/35; 0.035 mg, daily, for 3 of 4 weeks), or combined use of NAF and NOR 1/35 for 24 weeks. At baseline and every 8 weeks, we measured gonadotropins, estrogens, **androgens**, and **hair growth**. Bone density was assessed by dual energy x-ray adsorptiometry, and hot flashes were measured objectively. Baseline total **testosterone** (T), free T, percent free T, and sex hormone-binding globulin-binding capacity were similar among groups. With **treatment**, significant **reductions** (P = 0.01) in total T were seen with combination and NAF only **therapy**. Significant **increases** (P lt 0.001) in the sex hormone-binding globulin-binding capacity were seen in women given NOR 1/35 alone or in combination with NAF. Free T levels decreased to approximately half of baseline levels with combination **treatment** (17.9 to 6.4 nmol/L; P lt 0.001) and NOR 1/35 alone (20.8 to 10.2 nmol/L; P lt 0.001). There was a significant decrease in **hair** shaft diameter after combination **therapy** (P lt 0.05) that was not seen with either agent alone. Combination **therapy** also prevented the hot flashes and bone loss that occurred with agonist alone. In summary, our results demonstrate that combination GnRH agonist and low dose oral contraceptive **therapy** is more effective than either agent alone in the **treatment** of hirsutism and avoids the hypoestrogenic complications that occur with agonist only **therapy**.
ST RESEARCH ARTICLE; HUMAN; NAFARELIN; HORMONE-DRUG; ETHINYL ESTRADIOL; HORMONE-DRUG; NORETHINDRONE; HORMONE-DRUG; **TESTOSTERONE**; SEX HORMONE-BINDING GLOBULIN; HYPERANDROGENISM; HYPOESTROGENEMIA; **ANDROGEN**; **HAIR GROWTH** SUPPRESSION
RN 57-63-6 (ETHINYL ESTRADIOL)
58-22-0 (TESTOSTERONE)
68-22-4 (NORETHINDRONE)
76932-56-4 (NAFARELIN)
CC Biochemical Studies-General 10060
Biochemical Studies-Proteins, Peptides and Amino Acids 10064
Biochemical Studies-Sterols and Steroids 10067
Biophysics-Molecular Properties and Macromolecules *10506
Pathology, General and Miscellaneous-Therapy *12512
Metabolism-Sterols and Steroids *13008
Metabolism-Metabolic Disorders *13020
Endocrine System-Adrenals *17004
Endocrine System-Gonads and Placenta *17006
Endocrine System-Neuroendocrinology *17020
Integumentary System-Pathology *18506
Dental and Oral Biology-General; Methods *19001
Pharmacology-Clinical Pharmacology *22005
Pharmacology-Endocrine System *22016
Pharmacology-Reproductive System; Implantation Studies

***22028**
Routes of Immunization, Infection and Therapy *22100
Developmental Biology-Embryology-Morphogenesis, General *25508
BC **Hominidae 86215**

L130 ANSWER 31 OF 97 BIOSIS COPYRIGHT 1998 BIOSIS
AN 96:26272 BIOSIS
DN 98598407
TI Clinical efficacy and safety of low-dose flutamide alone and combined with an oral contraceptive for the **treatment** of idiopathic hirsutism.
AU Dodin S; Faure N; Cedrin I; Mechain C; Turcot-Lemay L; Guy J; Lemay A
CS Cent. Rech., Hopital St-Francois d'Assise, 10 rue de l'Espinay, Quebec, PQ G1L 3L5, Canada
SO Clinical Endocrinology 43 (5). 1995. 575-582. ISSN: 0300-0664
LA English
PR Biological Abstracts Vol. 101 Iss. 002 Ref. 026112
AB BACKGROUND AND OBJECTIVE: High doses of flutamide, which is the only antiandrogen that specifically blocks the **androgen** receptor, have recently been used with good clinical results in women with hirsutism. Since regression of **hair growth** requires long-term **therapy**, clinical and economic considerations are important. The use of the lowest efficacious dosage could **reduce** costs. This study was undertaken to compare safety and efficacy of a low dose of flutamide (125 mg twice daily) alone and in combination with a triphasic oral contraceptive (OC) in women with idiopathic hirsutism. PATIENTS: Flutamide was administered orally in a low dose of 125 mg twice daily for 12 months alone in women with no risk of pregnancy or during the use of an oral contraceptive. MEASUREMENTS: Women were seen every 3 months and were evaluated for hirsutism score, hormone and lipid measurements. DESIGN: The study, which was conducted as a prospective open trial, was proposed to patients with idiopathic hirsutism, that is, with serum **androgen** levels in normal range and LH/FSH ratio less than 2. RESULTS: A statistically significant decrease in hirsutism score as compared to baseline was observed after only 3 months with either **treatment**, flutamide alone (1 6.9 +- 1 .6 vs 14.2 +- 1.7, P lt 0.0001) or the combination of flutamide with OC (15.6 +- 0.8 vs 11 .9 +- 0.8, P lt 0.001). Three months after cessation of **treatment** a statistically significant decrease from baseline was observed in the two groups. Nevertheless, at 6 months post-**treatment** this decrease was still significant only in the group who took flutamide in combination with an oral contraceptive. Flutamide alone does not appear to modify the levels of lipoproteins. The association of flutamide with a triphasic formulation significantly **increased** the HDL-C levels. CONCLUSIONS: This study shows beneficial effects of a low dose of flutamide in women with idiopathic hirsutism. The addition of an oral contraceptive is judicious to prevent pregnancy and **reduce** recurrence of hirsutism after cessation of flutamide. Peripheral **androgenic** blockage does not modify lipid profiles and it might **reduce** the negative effect of oral contraceptive on HDL-C levels. The addition of electrolysis delays the recurrence of hirsutism after cessation of flutamide.
ST RESEARCH ARTICLE; HUMAN; FLUTAMIDE; ANTIANDROGEN; **ANDROGEN** RECEPTOR
RN 13311-84-7 (FLUTAMIDE)
CC Biochemical Studies-General 10060
Biochemical Studies-Sterols and Steroids 10067
Pathology, General and Miscellaneous-Therapy *12512
Endocrine System-Adrenals *17004
Integumentary System-Pathology *18506
Pharmacology-Clinical Pharmacology *22005
Pharmacology-Endocrine System *22016

BC **Hominidae 86215**

L130 ANSWER 32 OF 97 HCAPLUS COPYRIGHT 1998 ACS
AN 1995:562407 HCAPLUS
DN 123:4198
TI Characterization of recombinant human liver thermolabile phenol **sulfotransferase** with minoxidil as the substrate
AU Kudlacek, Patrick E.; Clemens, Dahn L.; Anderson, Robert J.
CS Section Endocrinology Metabolism, Creighton Univ. Sch. Med., Omaha, NE, 68105, USA
SO Biochem. Biophys. Res. Commun. (1995), 210(2), 363-9
CODEN: BBRCA9; ISSN: 0006-291X
DT Journal
LA English
CC 7-2 (Enzymes)
Section cross-reference(s): 1, 13
AB Minoxidil, a potent antihypertensive agent and **hair growth** stimulator, is metabolized by phenol **sulfotransferase** to its activated form, minoxidil sulfate. The thermostable form of phenol **sulfotransferase** was reported to be the enzyme that catalyzed the reaction. The previous findings with partially purified human platelet prepns. indicated that the thermolabile form of phenol **sulfotransferase** also catalyzed the sulfation of minoxidil. To confirm and to characterize precisely the activity of thermolabile phenol **sulfotransferase** toward minoxidil, the authors investigated the ability of the enzyme expressed from a human liver cDNA clone to sulfate minoxidil during testing of thermal stability and of inhibition of 2,6-dichloro-4-nitrophenol and NaCl. The cDNA encoded thermolabile phenol **sulfotransferase** activity assayed with minoxidil behaved in the same fashion as the activity measured with dopamine, a finding that confirmed that this enzyme activity sulfated minoxidil. Thus, thermolabile phenol **sulfotransferase** must be taken into account with the thermostable enzyme when estg. the human tissue **sulfotransferase** contribution to minoxidil sulfation.
ST phenol **sulfotransferase** minoxidil characterization
IT Liver
(characterization of recombinant human liver thermolabile phenol **sulfotransferase** with minoxidil as substrate)
IT 51-61-6, Dopamine, biological studies 9026-09-9, Phenol **sulfotransferase** 38304-91-5, Minoxidil
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(characterization of recombinant human liver thermolabile phenol **sulfotransferase** with minoxidil as substrate)
IT 83701-22-8, Minoxidil sulfate
RL: BPR (Biological process); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative); PROC (Process)
(characterization of recombinant human liver thermolabile phenol **sulfotransferase** with minoxidil as substrate)

L130 ANSWER 33 OF 97 BIOSIS COPYRIGHT 1998 BIOSIS DUPLICATE 6
AN 95:124110 BIOSIS
DN 98138410
TI Phenobarbital in **hair** and drug monitoring.
AU Goulle J P; Noyon J; Layet A; Rapoport N F; Vaschalde Y; Pignier Y; Bouige D; Jouen F
CS Centre Hospitalier, BP24, 76083 Le Havre cedex, France
SO Forensic Science International 70 (1-3). 1995. 191-202. ISSN: 0379-0738
LA English
PR Biological Abstracts Vol. 099 Iss. 007 Ref. 094967
AB Phenobarbital analysis was performed in vertex **hair** of

patients by gas chromatography mass spectrometry (GC/MS). After washing with dichloromethane, about 250 mg were ground to dust in a ball mill. A 50-mg sample was stirred mechanically for 10 min with 3 ml of NH-4Cl/HCl buffer (pH 2.0) containing phenobarbital D-5. A solid phase extraction was performed (extrelut Merck) and elution was achieved with chloroform/isopropanol/n-heptane (50:17:33; v/v). A full scan (40-240 uma) acquisition was realized by GC/MS with an ion trap (ITD 700 Finnigan) using a DB5-MS chromatographic column. Quantification was achieved by integrating dominants ions (phenobarbital, 204; phenobarbital D-5, 209). Compared to serum, **hair** concentrates phenobarbital during anti-epileptic therapy (average value 36.4 ng/mg, n = 40 vs. 18.7 mg/l, n = 23). A group correlation exists between phenobarbital in **hair** and phenobarbital in serum, and between phenobarbital in **hair** and clinic observation in some typical cases. Phenobarbital in **hair** yields good information over a long period, especially when blood collection has not been made, when clinical disorders are observed on long-term therapeutic observance.

ST RESEARCH ARTICLE; HUMAN; PHENOBARBITAL; ANTICONVULSANT-DRUG; BLOOD; SALIVA; URINE; FORENSICS; GAS CHROMATOGRAPHY; MASS SPECTROMETRY; ANALYTICAL METHOD

RN **50-06-6** (PHENOBARBITAL)

CC General Biology-Forensic Science *00531
Biochemical Studies-General 10060
Biophysics-General Biophysical Techniques 10504
Biophysics-Molecular Properties and Macromolecules 10506
Blood, Blood-Forming Organs and Body Fluids-Blood and Lymph Studies *15002
Blood, Blood-Forming Organs and Body Fluids-Other Body Fluids *15010
Urinary System and External Secretions-Physiology and Biochemistry *15504
Integumentary System-Physiology and Biochemistry *18504
Dental and Oral Biology-Physiology and Biochemistry *19004
Pharmacology-Clinical Pharmacology *22005
Pharmacology-Neuropharmacology *22024

BC **Hominidae 86215**

L130 ANSWER 34 OF 97 BIOSIS COPYRIGHT 1998 BIOSIS

AN 95:257466 BIOSIS

DN 98271766

TI Efficacy of low-dose GnRH analogue (Buserelin) in the **treatment** of hirsutism.

AU Bertoli A; Fusco A; Magnani A; Marini M A; Di Daniele N; Gatti S; Lauro R

CS Cattedra Endocrinol., Med. Interna, Dip. Univ. Roma, Via O. Raimondo, I-00173 Roma, Italy

SO Experimental and Clinical Endocrinology & Diabetes 103 (1). 1995. 15-20. ISSN: 0947-7349

LA English

PR Biological Abstracts Vol. 099 Iss. 012 Ref. 176684

AB The aim of the present study was to evaluate the effect of low dose GnRH analogue (Buserelin) on gonadal steroid secretion and **hair growth** in hirsute women. The drug was administered as a nasal spray (200 mu-g tid) to reduce gonadal steroid secretion. Eight hirsute women were **treated** for six month with the gonadotropin-releasing hormone analog. All had subclinical polycystic ovary syndromes on the basis of ultrasound or hormonal data, together with ovary dysfunctions and irregular menses. None had adrenal or pituitary dysfunction. The score of hirsutism was evaluated according to Ferriman and Gallway; pituitary function was evaluated measuring the FSH and LH response to GnRH **stimulation** and gonadal steroid secretion by measuring estradiol, progesterone, total plasma **testosterone**, androstenedione and androstanediol. Sex hormone binding globulin,

insulin, prolactin and DHEA-S were also measured. The suppression of ovarian steroid secretion was confirmed by reductions in total plasma **testosterone**, androstenedione and androstanediol that were detectable after one month of **treatment**. FSH and LH responses to GnRH **stimulation** were **inhibited** consistent with pituitary desensitization. No significant side effects were observed and all patients completed the trial. The score of hirsutism was 24 +- 5 before, 19.6 +- 6 by the 3rd month and 16.8 +- 5.1 by the 6th month of **treatment** (p lt 0.001); the effect was still evident 1 and 6 months after the withdrawal of the **therapy** (14.8 +- 3 and 15.8 +- 5 respectively; p lt 0.001). Our findings indicate that Buserelin is useful in the **treatment** of non adrenal hirsutism when other forms of **therapy** are contraindicated or poorly tolerated by the patient.

ST RESEARCH ARTICLE; HUMAN; BUSERELIN; DERMATOLOGICAL-DRUG; BUSERELIN; HORMONE-DRUG; BUSERELIN; METABOLIC-DRUG; GONADOTROPIN-RELEASING HORMONE; FSH; LUTEINIZING HORMONE; NON-ADRENAL HIRSUTISM; SUBCLINICAL POLYCYSTIC OVARY SYNDROME; OVARIAN DYSFUNCTION; IRREGULAR MENSES

RN 9002-67-9 (LUTEINIZING HORMONE)
9002-68-0 (FSH)
57982-77-1 (BUSERELIN)

CC Circadian Rhythms and Other Periodic Cycles *07200
Clinical Biochemistry; General Methods and Applications *10006
Biochemical Studies-Proteins, Peptides and Amino Acids 10064
Biochemical Studies-Sterols and Steroids 10067
Biochemical Studies-Carbohydrates 10068
Pathology, General and Miscellaneous-Therapy *12512
Metabolism-Sterols and Steroids *13008
Reproductive System-Physiology and Biochemistry *16504
Reproductive System-Pathology *16506
Endocrine System-Adrenals *17004
Endocrine System-Gonads and Placenta *17006
Endocrine System-Pituitary *17014
Endocrine System-Neuroendocrinology *17020
Integumentary System-Physiology and Biochemistry *18504
Integumentary System-Pathology *18506
Pharmacology-Drug Metabolism; Metabolic Stimulators *22003
Pharmacology-Clinical Pharmacology *22005
Pharmacology-Endocrine System *22016
Pharmacology-Integumentary System, Dental and Oral Biology *22020
Pharmacology-Neuropharmacology *22024

BC **Hominidae 86215**

L130 ANSWER 35 OF 97 WPIDS COPYRIGHT 1998 DERWENT INFORMATION LTD
AN 94-183112 [22] WPIDS
DNC C94-082933
TI Process of cosmetically **inhibiting** mammalian **hair growth** - comprising applying to the skin a compsn including an **inhibitor** comprising pantothenic acid or an analogue of pantothenic acid.
DC B05 D21 E16
IN AHLUWALIA, G S; SHANDER, D
PA (HAND-I) HANDELMAN J H; (AHLU-I) AHLUWALIA G S; (SHAN-I) SHANDER D
CYC 47
PI WO 9410967 A1 940526 (9422)* EN 17 pp A61K007-06 <--
RW: AT BE CH DE DK ES FR GB GR IE IT LU MC NL OA PT SE
W: AT AU BB BG BR BY CA CH CZ DE DK ES FI GB HU JP KP KR KZ LK LU LV MG MN MW NL NO NZ PL PT RO RU SD SE SK UA US UZ VN
AU 9455529 A 940608 (9435) A61K007-06 <--
US 5364885 A 941115 (9445) 5 pp A61K031-195 <--
EP 667766 A1 950823 (9538) EN A61K007-06 <--
R: AT BE CH DE DK ES FR GB GR IE IT LI NL PT SE

JP 08503220 W 960409 (9645) 12 pp A61K007-06 <--
EP 667766 B1 970813 (9737) EN 7 pp A61K007-06 <--
R: AT BE CH DE DK ES FR GB GR IE IT LI NL PT SE
DE 69313128 E 970918 (9743) A61K007-06 <--
ES 2107789 T3 971201 (9803) A61K007-06 <--
ADT WO 9410967 A1 WO 93-US10920 931110; AU 9455529 A AU 94-55529 931110; US 5364885 A US 92-976446 921113; EP 667766 A1 WO 93-US10920 931110, EP 94-900614 931110; JP 08503220 W WO 93-US10920 931110, JP 94-512359 931110; EP 667766 B1 WO 93-US10920 931110, EP 94-900614 931110; DE 69313128 E DE 93-613128 931110, WO 93-US10920 931110, EP 94-900614 931110; ES 2107789 T3 EP 94-900614 931110
FDT AU 9455529 A Based on WO 9410967; EP 667766 A1 Based on WO 9410967; JP 08503220 W Based on WO 9410967; EP 667766 B1 Based on WO 9410967; DE 69313128 E Based on EP 667766, Based on WO 9410967; ES 2107789 T3 Based on EP 667766
PRAI US 92-976446 921113
REP GB 1458349; WO 9114431
IC ICM **A61K007-06; A61K031-195**
ICS **A61K007-155; A61K031-16**
AB WO 9410967 A UPAB: 940722
A process of cosmetically **inhibiting** mammalian **hair growth** comprises applying to the skin a compsn. including an **inhibitor** comprising pantothenic acid or an analogue of pantothenic acid.
USE/ADVANTAGE - The compsn. may be applied to skin on the face, neck, leg, arm, torso or armpit of the mammal; it is suitable for **inhibiting** human **hair growth**.
Pantothenic acid has been previously used in hair treatment methods. However, previous methods have focused on the use of pantothenic acid as a hair moisturiser and stimulant of scalp **hair growth**.
Dwg.0/0
FS CPI
FA AB; DCN
MC CPI: B10-C04D; B14-R02; D08-B03; E10-C04D5; E10-D03C

L130 ANSWER 36 OF 97 BIOSIS COPYRIGHT 1998 BIOSIS
AN 95:82835 BIOSIS
DN 98097135
TI Sequential estrogen-progestin addition to gonadotropin-releasing hormone agonist suppression for the chronic **treatment** of ovarian hyperandrogenism: A pilot study.
AU Lemay A; Faure N
CS Hopital St-Francois d'Asise, 10 rue de l'Espinay, Quebec, PQ G1L 3L5, Canada
SO Journal of Clinical Endocrinology & Metabolism 79 (6). 1994. 1716-1722. ISSN: 0021-972X
LA English
PR Biological Abstracts Vol. 099 Iss. 005 Ref. 067545
AB The purpose of the study was to evaluate the efficacy and safety of a sequential regimen of estrogen-progestin addition to GnRH agonist suppression in ovarian hyperandrogenism. Eight patients presenting with a polycystic ovary syndrome were **treated** with an se implant of GnRH agonist every 4 weeks for 48 weeks. Starting at week 9, patients were replaced with 100 mu-g transdermal estradiol patches continuously and sequentially combined with 10 mg oral medroxyprogesterone acetate the last 2 weeks of each 4-week period. The rapid down-regulation of the pituitary-ovarian axis led to significant **reduction** of **testosterone** and androstenedione to 48.9% and 67.4% of baseline, respectively. During steroid replacement, **testosterone** and androstenedione continued to decrease gradually. The baseline hirsutism score (18.7 +- 1.3) progressively fell to 9.7 +- 2.0 at the end of **treatment**. The mean **hair** diameter was significantly

reduced (0.097 +- 0.004 vs. 0.081 +- 0.005 mm). A withdrawal bleeding was obtained in 63.6% of the artificial cycles, but breakthrough bleeding occurred during 48% of the sequential replacements. The incidence of menopausal symptoms was low. There was a nonsignificant decrease in bone mineral content of the lumbar spine and the femoral neck but no trend in Ca-2+/creatinine and OH-proline (OH-P)/creatinine ratios or in serum triglycerides and cholesterol fractions. There was a nonsignificant **increase** in hirsutism score in five patients followed up for 24 weeks after cessation of **treatment**, although there was a rapid return of hormones toward baseline and recurrence of irregular bleeding. Transdermal estradiol addition periodically combined with medroxyprogesterone acetate is effective in **reducing** hirsutism and is safe in minimizing side effects and bone loss. A regimen allowing a better bleeding control would make this approach a valuable alternative for prolonged or repeated palliative **treatment** of excessive **hair growth** and irregular bleeding in polycystic ovary syndrome.

ST RESEARCH ARTICLE; HUMAN; ESTROGEN-PROGESTIN; HORMONE-DRUG; GONADOTROPIN-RELEASING HORMONE AGONIST; HORMONE-DRUG; HIRSUTISM

CC Biochemical Studies-Proteins, Peptides and Amino Acids 10064
Biochemical Studies-Sterols and Steroids 10067
Pathology, General and Miscellaneous-Therapy 12512
Reproductive System-Pathology *16506
Endocrine System-Gonads and Placenta *17006
Endocrine System-Neuroendocrinology *17020
Integumentary System-Pathology *18506
Pharmacology-Clinical Pharmacology *22005
Pharmacology-Endocrine System *22016
Pharmacology-Reproductive System; Implantation Studies *22028

BC **Hominidae 86215**

L130 ANSWER 37 OF 97 BIOSIS COPYRIGHT 1998 BIOSIS

AN 94:551162 BIOSIS

DN 98010710

TI Clinical and hormonal effects of the 5-alpha-**reductase** **inhibitor** finasteride in idiopathic hirsutism.

AU Moghetti P; Castello R; Magnani C M; Tosi F; Negri C; Armanini D; Bellotti G; Muggeo M

CS Cattedra Malattie del Metabolismo, Ospedale Policlinico, I-37134 Verona, Italy

SO Journal of Clinical Endocrinology & Metabolism 79 (4). 1994. 1115-1121. ISSN: 0021-972X

LA English

PR Biological Abstracts Vol. 099 Iss. 001 Ref. 010710

AB Hyperactivity of 5-alpha-**reductase** in the skin is considered a major mechanism of excessive **hair growth** in hirsute women with normal levels of serum **androgens** (idiopathic hirsutism). Preventing the conversion of **testosterone** to dihydrotestosterone by **inhibiting** 5-alpha-**reductase** activity could thus be the most rational and effective **treatment** in this condition. The present study evaluated the effects of the oral administration of finasteride (5 mg once daily) for 6 months in 17 young women with idiopathic hirsutism, 5 of whom were also given an oral contraceptive. The degree of hirsutism (graded by a modified Ferriman-Gallwey score), serum sex hormone levels, and serum and urinary 5-alpha-metabolism steroid profiles were determined basally and periodically during the **treatment** period. The modified Ferriman-Gallwey score showed a remarkable **reduction** after 6 months of finasteride **treatment** (5.9 +- 0.6 us. 11.7 +- 1.3; P lt 0.01). Serum 5-alpha-dihydrotestosterone and 3a-androstanediol glucuronide levels were decreased, and urinary C-19

and C-21 5-beta/5-alpha metabolite ratios were **increased** compared with pretreatment values. No significant adverse effect was reported. In women **treated** with finasteride and oral contraceptive, clinical efficacy was slightly more pronounced. In conclusion, the 5-alpha-**reductase inhibitor** finasteride is well tolerated and seems to be a useful toot in the **treatment** of idiopathic hirsutism.

ST RESEARCH ARTICLE; WOMEN; FINASTERIDE; DERMATOLOGICAL-DRUG; ENZYME **INHIBITOR**-DRUG; HORMONE-DRUG; **TESTOSTERONE** CONVERSION PREVENTION; CLINICAL ENDOCRINOLOGY

RN 58-22-0 (TESTOSTERONE)
98319-26-7 (FINASTERIDE)

CC Biochemical Studies-Proteins, Peptides and Amino Acids 10064
Biochemical Studies-Sterols and Steroids 10067
Enzymes-Physiological Studies *10808
Pathology, General and Miscellaneous-Therapy *12512
Metabolism-Sterols and Steroids *13008
Endocrine System-Adrenals *17004
Integumentary System-Pathology *18506
Pharmacology-Clinical Pharmacology *22005
Pharmacology-Endocrine System *22016
Pharmacology-Integumentary System, Dental and Oral Biology *22020

BC **Hominidae 86215**

L130 ANSWER 38 OF 97 BIOSIS COPYRIGHT 1998 BIOSIS

AN 94:362128 BIOSIS

DN 97375128

TI Progressively intractable seizures, focal alopecia, and hemimegalencephaly.

AU Pelayo R; Barasch E; Kang H; Marion R; Moshe S L

CS Montefiore Med. Cent., NW7 EEG, 111 East 210th St., Bronx, NY 10467, USA

SO Neurology 44 (5). 1994. 969-971. ISSN: 0028-3878

LA English

PR Biological Abstracts Vol. 098 Iss. 005 Ref. 063617

AB We report a 3-year-old boy with the neurocutaneous combination of unilateral alopecia, ipsilateral hemimegalencephaly, and intractable seizures. He was born with an asymmetric **hair** pattern consisting of absent patches of **hair**, a small left eyebrow, and less eyelashes on the left eye; he had normal development until age 17 months, when the experienced right focal seizures with fever. Two months later, fever triggered new seizures characterized by flurries of head and body flexion and adduction of the right arm. He had left hand preference and language regression. EEG manifested left hemihypsarrhythmia, and MRI showed left hemimegalencephaly with marked enlargement of the temporal lobe with ventriculomegaly. Seizures were refractory to treatment with phenobarbital, adrenocorticotropic hormone, pyridoxine, sodium valproate, clonazepam, carbamazepine, phenytoin, and felbamate. This may represent a previously undescribed neurocutaneous syndrome.

ST CASE STUDY; HUMAN; CHILD; PHENOBARBITAL; ANTICONVULSANT-DRUG; PYRIDOXINE; ANTICONVULSANT-DRUG; SODIUM VALPROATE; ANTI - CONVULSANT DRUG; CLONAZEPAM; ANTICONVULSANT-DRUG; CARBAMAZEPINE; ANTICONVULSANT-DRUG; PHENYTOIN; ANTICONVULSANT-DRUG; FELBAMATE; ANTICONVULSANT-DRUG; ACTH; FEVER; LANGUAGE REGRESSION; MAGNETIC RESONANCE IMAGING; ELECTROENCEPHALOGRAM

RN **50-06-6** (PHENOBARBITAL)
57-41-0 (PHENYTOIN)
65-23-6 (PYRIDOXINE)
298-46-4 (CARBAMAZEPINE)
1069-66-5 (SODIUM VALPROATE)
1622-61-3 (CLONAZEPAM)
9002-60-2 (ACTH)

25451-15-4 (FELBAMATE)
CC Genetics and Cytogenetics-Human *03508
Biochemical Studies-General 10060
Biochemical Studies-Proteins, Peptides and Amino Acids 10064
Anatomy and Histology, General and Comparative-Radiologic Anatomy 11106
Chordate Body Regions-Head 11304
Endocrine System-Pituitary *17014
Integumentary System-Pathology *18506
Sense Organs, Associated Structures and Functions-Physiology and Biochemistry *20004
Sense Organs, Associated Structures and Functions-Deafness, Speech and Hearing *20008
Nervous System-Pathology *20506
Psychiatry-Mental Retardation *21006
Temperature: Its Measurement, Effects and Regulation-Thermopathology *23007
Pediatrics *25000
Developmental Biology-Embryology-Descriptive Teratology and Teratogenesis *25552
BC **Hominidae 86215**

L130 ANSWER 39 OF 97 BIOSIS COPYRIGHT 1998 BIOSIS
AN 94:491290 BIOSIS
DN 97504290
TI Effects of Finasteride, a 5-alpha-**Reductase Inhibitor**, on Circulating **Androgens** and Gonadotropin Secretion in Hirsute Women.
AU Fruzzetti F; De Lorenzo D; Parrini D; Ricci C
CS Clin. Ostet. Ginecol., Univ. degli Studi Pisa, Via Roma 35, 56100 Pisa, ITL
SO Journal of Clinical Endocrinology & Metabolism 79 (3). 1994. 831-835. ISSN: 0021-972X
LA English
PR Biological Abstracts Vol. 098 Iss. 010 Ref. 140029
AB An oral 5-mg dose of finasteride, a 5-alpha-**reductase inhibitor**, was administered for 3 months to 10 hirsute women to determine the effect on gonadotropin secretion, on basal and **stimulated androgen** secretion, and on **hair growth**. **Hair growth** was assessed by the Ferriman-Gallwey score. All of the above determinations were evaluated before and after 1 and/or 3 months of finasteride **treatment**. Basal and GnRH-**stimulated** gonadotropin secretions were not affected. Indeed, finasteride did not modify the pulsatility of LH secretion. No change was seen in estradiol, PRL, free **testosterone**, androstenedione, dehydroepiandrosterone sulfate, and sex hormone-binding globulin concentrations. Serum concentrations of cortisol (F) were significantly **reduced** after 1 month of finasteride **treatment**. The F levels returned to pretreatment levels after 3 months. Plasma levels of dihydrotestosterone and 3-alpha-androstanediol glucuronide significantly decreased during finasteride **treatment**. A significant **increase** in **testosterone** concentrations was observed after 3 months. Finasteride did not modify the responses of **testosterone**, androstenedione, and dehydroepiandrosterone sulfate to ACTH-(1-24) injection. Conversely, finasteride blunted the F response to corticotropin **stimulation**. Three months of finasteride **treatment** significantly decreased the Ferriman-Gallwey score. In conclusion, finasteride significantly decreased dihydrotestosterone and **hair growth** in hirsute women without negatively affecting gonadotropin secretion.
ST RESEARCH ARTICLE; FINASTERIDE; ENZYME **INHIBITOR**-DRUG; HORMONE-DRUG; LUTEINIZING HORMONE METABOLISM; DIHYDROTESTOSTERONE

DECREASE; **HAIR GROWTH REDUCTION;**
THERAPEUTIC METHOD
RN 521-18-6 (DIHYDROTESTOSTERONE)
9002-67-9 (LUTEINIZING HORMONE)
98319-26-7 (FINASTERIDE)
CC Genetics and Cytogenetics-Human *03508
Biochemical Studies-Proteins, Peptides and Amino Acids 10064
Biochemical Studies-Sterols and Steroids 10067
Biochemical Studies-Carbohydrates 10068
Enzymes-Chemical and Physical *10806
Enzymes-Physiological Studies *10808
Metabolism-Sterols and Steroids *13008
Reproductive System-Pathology *16506
Endocrine System-Adrenals *17004
Endocrine System-Pituitary *17014
Integumentary System-Physiology and Biochemistry *18504
Pharmacology-Drug Metabolism; Metabolic Stimulators *22003
Pharmacology-Clinical Pharmacology *22005
Pharmacology-Endocrine System *22016
BC **Hominidae 86215**

L130 ANSWER 40 OF 97 BIOSIS COPYRIGHT 1998 BIOSIS
AN 94:465397 BIOSIS
DN 97478397
TI **Hair**: A potential biomarker for drug and chemical exposure.
AU Wilkins D; Gygi S; Haughey H; Rollins D
CS Cent. Human Toxicol., Univ. Utah, Salt Lake City, UT 84108, USA
SO North American Congress of Clinical Toxicology-94, Salt Lake City, Utah, USA, September 22-26, 1994. Veterinary and Human Toxicology 36 (4). 1994. 341. ISSN: 0145-6296
DT Conference
LA English
PR Biological Abstracts/RRM Vol. 046 Iss. 011 Ref. 176260
ST MEETING ABSTRACT; MEETING POSTER; HUMAN; CODEINE; PHENOBARBITAL; **HAIR** BULBS; DISTAL **HAIR** SEGMENTS
RN **50-06-6** (PHENOBARBITAL)
76-57-3 (CODEINE)
CC General Biology-Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals 00520
Clinical Biochemistry; General Methods and Applications *10006
Biochemical Studies-General 10060
Metabolism-General Metabolism; Metabolic Pathways *13002
Integumentary System-Physiology and Biochemistry *18504
Pharmacology-General *22002
Pharmacology-Clinical Pharmacology *22005
Toxicology-General; Methods and Experimental *22501
BC **Hominidae 86215**

L130 ANSWER 41 OF 97 HCAPLUS COPYRIGHT 1998 ACS
AN 1994:645044 HCAPLUS
DN 121:245044
TI Minoxidil sulfation in the **hair** follicle
AU Baker, C.A.; Uno, H.; Johnson, G.A.
CS Upjohn Company, Kalamazoo, MI, USA
SO Skin Pharmacol. (1994), 7(6), 335-9
CODEN: SKPHEU; ISSN: 1011-0283
DT Journal
LA English
CC 1-2 (Pharmacology)
AB The in vivo model which may be the most accurate for the ability to predict **hair growth** in humans, and which was utilized in the preclin. development of minoxidil, is the adult stump-tailed macaque. Previous reports have suggested that the enzyme activity which accounts for the activation of minoxidil,

i.e., minoxidil **sulfotransferase**, is present in skin. We have demonstrated that scalp skin from the stump-tailed macaque contains minoxidil **sulfotransferase** activity, and further with dissection of that scalp skin into epidermis, dermis and **hair** follicle, most of **sulfotransferase** activity was present in the follicle. **Sulfotransferase** activity in the **hair** follicle in freeze-dried scalp skin sections from 9 stump-tailed macaques ranged from 47 to 84% of the total (mean 61 .+-. 12%). Much less minoxidil **sulfotransferase** activity was measured in the epidermis (mean 18 .+-. 11%, with a range of 2-37%) and the dermis (mean 21 .+-. 8%, with a range of 4-35%) of these scalp sections. These results indicate that the scalp skin from the stump-tailed macaque contains minoxidil **sulfotransferase** activity and this activity is largely localized in the **hair** follicle which may account for its ability to stimulate **hair growth** in this animal model.

ST minoxidil **sulfotransferase hair** follicle macaque

IT Macaca
(**minoxidilsulfotransferase** activity in **hair** follicle of stump-tailed macaque)

IT **Hair**
(follicle, **minoxidilsulfotransferase** activity in **hair** follicle of macaque)

IT 38304-91-5, Minoxidil
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(minoxidil sulfation in **hair** follicle of macaque)

IT 129924-25-0, Minoxidil **sulfotransferase**
RL: BAC (Biological activity or effector, except adverse); BIOL (Biological study)
(**minoxidilsulfotransferase** activity in **hair** follicle of macaque)

L130 ANSWER 42 OF 97 BIOSIS COPYRIGHT 1998 BIOSIS
AN 94:454513 BIOSIS
DN 97467513
TI **Increased** cAMP levels in human **hair** follicles due to local **treatment** with trichoriboside.
AU Gazzani G; Roncoroni S; Corsi M; Falchi M; Ferrero M E
CS Istituto di Patologia Generale 31, Via Mangiagalli, 20133 Milano, ITL
SO International Journal of Tissue Reactions 16 (2). 1994. 73-77. ISSN: 0250-0868
LA English
PR Biological Abstracts Vol. 098 Iss. 009 Ref. 121825
AB Local **therapy** with trichoriboside and trichosaccharide, which have been found to be beneficial for scalp **hair** maintenance in adult males affected by **androgenic** alopecia, was found to **increase** cAMP levels in human scalp **hair** follicles. The **increase** was significant in men affected by **androgenic** alopecia, whereas it was not significant in unaffected control men. Trichoriboside showed a greater activity than trichosaccharide, and such activity was accompanied by a significant concomitant **reduction** of ATP in the **hair**.
ST RESEARCH ARTICLE; TRICHORIBOSIDE; DERMATOLOGICAL-DRUG; TRICHOSACCHARIDE; DERMATOLOGICAL-DRUG; CYCLIC AMP; ATP; **ANDROGENIC** ALOPECIA; **HAIR GROWTH**
RN 60-92-4 (CYCLIC AMP)
113552-93-5 (TRICHOSACCHARIDE)
56-65-5Q, 87805-51-4Q, 94587-45-8Q, 111839-44-2Q (ATP)
CC Biochemical Studies-General 10060
Biochemical Studies-Nucleic Acids, Purines and Pyrimidines 10062
Biochemical Studies-Carbohydrates 10068

Pathology, General and Miscellaneous-Therapy *12512
Metabolism-Nucleic Acids, Purines and Pyrimidines *13014
Integumentary System-Pathology *18506
Pharmacology-Clinical Pharmacology *22005
Pharmacology-Integumentary System, Dental and Oral Biology *22020
Developmental Biology-Embryology-Morphogenesis, General *25508
BC **Hominidae 86215**

L130 ANSWER 43 OF 97 WPIDS COPYRIGHT 1998 DERWENT INFORMATION LTD
AN 93-167280 [20] WPIDS
CR 95-350220 [45]
DNC C93-074552
TI **Redn.** of **hair growth** and altering character - by topical application of L-asparagine synthetase **inhibitor** e.g. guanidino-succinic acid.
DC B05 D21 E19 P14
IN AHLUWALIA, G S; HANDELMAN, J H
PA (HAND-I) HANDELMAN J H
CYC 39
PI WO 9308687 A1 930513 (9320)* EN 9 pp A01N037-10
RW: AT BE CH DE DK ES FR GB GR IE IT LU MC NL OA SE
W: AT AU BB BG BR CA CH CS DE DK ES FI GB HU JP KP KR LK LU MG MN MW NL NO PL RO RU SD SE UA US
AU 9230627 A 930607 (9338) A01N037-10
EP 612211 A1 940831 (9433) EN A01N037-10
R: AT BE CH DE DK ES FR GB GR IE IT LI NL SE
JP 07504646 W 950525 (9529) A61K031-19 <--
EP 612211 A4 941207 (9542) A01N037-10
AU 670554 B 960725 (9637) A61K031-19 <--
CA 2122002 C 971216 (9810) A61K007-06 <--
ADT WO 9308687 A1 WO 92-US9438 921104; AU 9230627 A AU 92-30627 921104; EP 612211 A1 EP 92-924244 921104, WO 92-US9438 921104; JP 07504646 W WO 92-US9438 921104, JP 93-508679 921104; EP 612211 A4 EP 92-924244 ; AU 670554 B AU 92-30627 921104; CA 2122002 C CA 92-2122002 921104
FDT AU 9230627 A Based on WO 9308687; EP 612211 A1 Based on WO 9308687; JP 07504646 W Based on WO 9308687; AU 670554 B Previous Publ. AU 9230627, Based on WO 9308687
PRAI US 91-788168 911105
REP US 4435419; 2.Jnl.Ref
IC ICM A01N037-10; **A61K007-06**; **A61K031-19**
ICS A01K067-00; A01N037-12; **A61K031-195**
AB WO 9308687 A UPAB: 951122
Redn. of rate and altering character of mammalian **hair growth**, comprising application of a compsn. contg. an organic **inhibitor** of L-aspargine synthetase, is new.
Inhibitors are pref. guandinosuccinic acid, oxaloacetic acid, cysteinesulphinic acid, diethyl aminomalonate, or ethacrynic acid.
USE - The **inhibitor** is non-irritant, as inorganic materials are. It affects partic. **androgen stimulated hair growth.** Compsns. comprise 0.1-30% **inhibitor** and opt. a penetration enhancer, and the application rate is 10-7500 mcg/sq.cm. of skin
Dwg.0/0
Dwg.0/0
FS CPI GMPI
FA AB; DCN
MC CPI: B10-A17; B12-G01B6; B12-L05; D08-B03; E10-A09C; E10-A17; E10-B02D5; E10-C02F; E10-C03

L130 ANSWER 44 OF 97 WPIDS COPYRIGHT 1998 DERWENT INFORMATION LTD
AN 93-267042 [34] WPIDS

DNN N93-204880 DNC C93-118985
TI Evaluation of hair tonic - by depilating backs of mice, painting with **testosterone** soln., then painting with the hair tonic.
DC D21 J04 S03
PA (NOEV-N) NOEVIR KK
CYC 1
PI JP 05180828 A 930723 (9334)* 4 pp G01N033-15
ADT JP 05180828 A JP 91-359786 911227
PRAI JP 91-359786 911227
IC ICM G01N033-15
ICS **A61K007-06**
AB JP05180828 A UPAB: 931119
Back regions of mice are depilated. **Testosterone** soln. is painted continuously to prolong resting phase of the hair follicle. Painting of the **testosterone** soln. is stopped to control transfer to **growth** phase of the **hair** follicle at the same time. Hair tonic is then painted for evaluation. The **testosterone** soln. is a 5 wt.% alcoholic soln..

USE/ADVANTAGE - At the initiation stage from resting phase to growth phase of follicle of mice, evaluation of hair tonic can be initiated with test samples and control under the same conditions. Variation of evaluation results is **reduced** and reproducibility of the evaluation **increased.**

In an example back regions of C3H mice were depilated. 5 wt.% **testosterone** ethanol soln. was painted once a day for 7-10 days continuously. One painting amt. was 0.3-0.5 ml. Painting of hair tonic was initiated, and trichogenous state compared with that of control (e.g. ethanol). Hair follicle were observed by HE staining. The hair tonics used were 2.0 wt.% hot extract or rosemary extract-contg. soln..

Dwg.0/2
FS CPI EPI
FA AB
MC CPI: D08-B03; J04-C04
EPI: S03-E14A1

L130 ANSWER 45 OF 97 WPIDS COPYRIGHT 1998 DERWENT INFORMATION LTD
AN 93-338052 [43] WPIDS
DNC C93-149502
TI Topical compsn. for treating the scalp contains cyproterone acetate - to **reduce hair** loss and **stimulate hair growth**, esp. in post menopausal women.
DC B05 D21 E15
IN UPHAUS, W; ZINGRAF, I
PA (ZING-I) ZINGRAF I
CYC 17
PI EP 566979 A1 931027 (9343)* DE 10 pp A61K007-06 <--
R: AT BE CH DE DK ES FR GB GR IE IT LI LU MC NL PT SE
DE 4213314 A1 931028 (9344) 7 pp A61K007-06 <--
ADT EP 566979 A1 EP 93-106092 930415; DE 4213314 A1 DE 92-4213314 920423
PRAI DE 92-4213314 920423
REP 2.Jnl.Ref ; DE 2840144; DE 3615396; DE 3621757; EP 163490; JP 61018711; JP 62103005; US 4684635; WO 8601402
IC ICM **A61K007-06**
ICS **A61K007-48; A61K031-57**
AB EP 566979 A UPAB: 931207
Compsn. for topical application to the scalp contains, apart from carriers and additives such as water, EtOH, castor oil and/or benzyl benzoate, and alcoholic soln. of cyproterone acetate (I) as active ingredient.

Pref. (I) is present at 0.01-1 wt.%.

For application to all of the scalp, the max. total application is 30ml 0.1% (I) soln. per week, and the starting application

20-30ml of 0.05% (I) soln.. If daily applications are made, the max. dose is 120ml of 0.025% soln. per week. Applications can also be made to only partic. regions of the scalp and women who are taking hormones should use the lotion only between days 4 and 21 of their menstrual cycle.

A pref. compsn. comprises 0.1g (I); 0.354g castor oil; 0.619g benzyl benzoate and 96% EtOH to make 100g.

USE/ADVANTAGE - The compsn. **reduces hair** loss and **stimulates hair growth** in all forms of **hair** loss of (partially) **androgenetic** origin, partic. in (post) menopausal women. (I) is a known **antiandrogen**, it is resorbed percutaneously so blocks the **androgen** receptors of the scalp without (at the doses used) causing any of the side effects associated with oral or parenteral admin.. The compsn. is applied at least twice a week, massaged in, then the hair covered for 30 min. with an occlusive bandage to prevent exposure to the air (this is necessary for good percutaneous resorption).

Dwg.0/4

FS CPI
FA AB; DCN
MC CPI: B01-C06; B12-G01A; B12-L05; D08-B03; E01

L130 ANSWER 46 OF 97 WPIDS COPYRIGHT 1998 DERWENT INFORMATION LTD
AN 93-010769 [02] WPIDS
DNC C93-004849
TI **Hair** loss and **hair growth** **stimulator** compsn. - contg. new 2,4-di amino pyrimidine 3-oxide derivs., useful for hair loss, alopecia, and desquamating dermatitis.
DC B03 D21 E13
IN GALEY, J; HOCQUAUX, M; MAIGNAN, J; TERRANOVA, E; TULOUP, R; TULUOP, R; GALEY, J B
PA (OREA) L'OREAL SA
CYC 17
PI EP 522964 A1 930113 (9302)* FR 29 pp C07D239-48
R: AT BE CH DE DK ES FR GB GR IT LI NL PT SE
FR 2678929 A1 930115 (9311) 37 pp C07D239-48
CA 2073755 A 930112 (9313) FR A61K007-06 <--
JP 05194230 A 930803 (9335) 19 pp A61K031-505 <--
US 5466694 A 951114 (9551) 15 pp A61K009-10 <--
ADT EP 522964 A1 EP 92-401980 920709; FR 2678929 A1 FR 91-8764 910711; CA 2073755 A CA 92-2073755 920713; JP 05194230 A JP 92-184089 920710; US 5466694 A US 92-912512 920713
PRAI FR 91-8764 910711
REP 2.Jnl.Ref ; DE 1695969; EP 356271
IC ICM **A61K007-06; A61K009-10; A61K031-505** ; C07D239-48
ICS **A61K009-06**; C07D239-46; C07D239-50
AB EP 522964 A UPAB: 931118
Compsn. contains in a physiologically acceptable medium cpd(s) of formula (I) or their acid salts. R1 and R3 are H; R2 and R4 are H or 1-4C alkyl; R5 is H, 1-12C alkyl, 3-12C alkenyl, 3-8C cycloalkyl, aryl, arylalkyl, hydroxyalkyl or aminoalkyl with 1-6C alkyl; X is H, halogen, 1-6C alkyl, NO2, benzoyloxy or -NHR6 (R6=H, acyl or 1-8C alkyl). Z is S or O; provided that Z is S when X is H or when R5 is aryl. Y is O or OSO3. Cpds (I) and their acid salts are claimed per se, except 2,4-diamino 6-hydroxy 5-bromopyrimidine 3-oxide; 2,4-diamino 6-thiophenyl, pyrimidine 3-oxide and their acid salts.

Prefd. (I) is 2,4-diamino 5-chloro 6-n-butyloxypyrimidine 3-oxide or 2,4-diamino 5-nitro 6-n-butyloxypyrimidine 3-oxide pharmaceutical compsns. contain 0.1-10 wt% of (I) and may be in the form of eg ointment, cream, powder, emulsion, imbibed pad and spray. Cosmetic compsns. contain 0.01-5 wt% of (I) and may be in the form

of lotion, gel, soap, shampoo and aerosol. The compsns. also contain hydrating agents, antiseborrhoeic agents, activators for (I) (eg nicotinic acid esters, (non)steroidal anti-inflammatory agents, retinoids, diazoxide, spiroxasone, phospholipids, lactones, and carotenoids), and surfactants.

USE/ADVANTAGE - (I) are used for the prepn. of a medicament for treating alopecia, hair loss and desquamating dermatitis, and for pharmaceutical or cosmetic compsn. for topical application. (I) are soluble in media usually used in cosmetic and pharmacy.

0/0

Dwg.0/0

FS CPI
FA AB; GI; DCN
MC CPI: B07-D12; B12-A07; B12-L02; B12-L05; D08-B03; E07-D12

L130 ANSWER 47 OF 97 HCAPLUS COPYRIGHT 1998 ACS
AN 1993:204673 HCAPLUS
DN 118:204673
TI Sulfate conjugation of minoxidil in rat skin
AU Wong, K. O.; Tan, Alex Y. H.; Lim, B. G.; Wong, Kim Ping
CS Fac. Med., Natl. Univ. Singapore, Singapore, 0511, Singapore
SO Biochem. Pharmacol. (1993), 45(5), 1180-2
CODEN: BCPCA6; ISSN: 0006-2952
DT Journal
LA English
CC 1-2 (Pharmacology)
AB Minoxidil **sulfotransferase** (MST) activity was detd. in the cytosolic fraction of rat skin and liver. MST of rat skin is similar to the P (phenol)-form of **phenosulfotransferase** (PST) of human tissues with respect to thermostability and inhibition by 2,6-dichloro-4-nitrophenol (DCNP). p-Nitrophenol, a prototype substrate of human P-PST form, inhibits MST at micromolar concn. while millimolar concns. of dopamine and tyramine, substrates of human M-(monoamine)-PST, are required to elicit a similar degree of inhibition. The enzymic transfer of 35S from sodium 35sulfate to minoxidil was also demonstrated, suggesting that the rat skin is potentially capable of synthesizing 3'-phosphoadenosine-5'-phosphosulfate (PAPS) from inorg. sulfate and utilizing it for the biosynthesis of minoxidil sulfate, its active metabolite. Thus, it is conceivable that the pharmacol. action of minoxidil as a promoter of **hair growth** could be carried out by the cutaneous tissues without the contribution of hepatic or other extrahepatic organs.
ST minoxidil sulfate conjugation skin
IT Liver, metabolism
Skin, metabolism
(sulfate conjugation of minoxidil in)
IT Cytoplasm
(cytosol, minoxidil **sulfotransferase** of, of liver and skin, minoxidil metab. by)
IT 83701-22-8, Minoxidil sulfate
RL: FORM (Formation, nonpreparative)
(formation of, from minoxidil, in skin and liver)
IT 129924-25-0, Minoxidil **sulfotransferase**
RL: BIOL (Biological study)
(of liver and skin, in sulfate conjugation of minoxidil)
IT 38304-91-5, Minoxidil
RL: PRP (Properties)
(sulfate conjugation of, in skin and liver)

L130 ANSWER 48 OF 97 HCAPLUS COPYRIGHT 1998 ACS
AN 1993:160404 HCAPLUS
DN 118:160404
TI Enzymic and nonenzymic sulfation mechanisms in the biological

actions of minoxidil
AU Meisheri, Kaushik D.; Johnson, Garland A.; Puddington, Lynn
CS Upjohn Lab., Upjohn Co., Kalamazoo, MI, 49001, USA
SO Biochem. Pharmacol. (1993), 45(2), 271-9
CODEN: BCPCA6; ISSN: 0006-2952
DT Journal; General Review
LA English
CC 1-0 (Pharmacology)
AB A review with 43 refs. An anal. of the scientific literature regarding minoxidil suggests that serendipitous observations coupled with exptl. pursuit of these observations by a small no. of investigators having played important roles during the discovery and development of minoxidil as an antihypertensive as well as a **hair growth** promoting agent. This is also true for the work done subsequently towards defining the cellular mechanism of action of minoxidil. This review will describe some of the salient features of the discovery of minoxidil as a unique drug entity, and will illustrate how this compd. has become a valuable tool for exposing some unique functional capacities of cells. These include identification of a **sulfotransferase** enzyme responsible for bioactivation of minoxidil, identification of a K+ channel opening mechanism for vasodilation, and identification of protein substrates for post-translational non-enzymic sulfate addn.
ST review minoxidil antihypertensive **hair growth** sulfation
IT **Hair**
(**growth** of, minoxidil promotion of, sulfation in, in humans and lab. animals)
IT Antihypertensives
(minoxidil as, sulfation in, in humans and lab. animals)
IT 38304-91-5, Minoxidil
RL: BIOL (Biological study)
(as antihypertensive and **hair growth** promotion by, sulfation in, in humans and lab. animals)
IT 9023-09-0, **Sulfotransferase**
RL: BIOL (Biological study)
(in antihypertensive and **hair growth** -promoting actions of minoxidil, in humans and lab. animals)

L130 ANSWER 49 OF 97 HCAPLUS COPYRIGHT 1998 ACS
AN 1992:490319 HCAPLUS
DN 117:90319
TI a process for the preparation of 5-fluoro-6-(1-piperidinyl)-2,4-pyrimidinediamine 3-oxide (5-fluorominoxidil) and is use as **hair growth** agent and antihypertensive
IN Schostarez, Heinrich Josef
PA Upjohn Co., USA
SO PCT Int. Appl., 19 pp.
CODEN: PIXXD2
PI WO 9208705 A1 920529
DS W: AU, BB, BG, BR, CA, CS, FI, HU, JP, KP, KR, LK, MC, MG, MN, MW, NO, PL, RO, SD, SU, US
RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, DE, DK, ES, FR, GA, GB, GR, IT, LU, ML, MR, NL, SE, SN, TD, TG
AI WO 91-US6728 910920
PRAI US 90-612695 901114
DT Patent
LA English
IC ICM C07D239-50
ICS A61K031-505; A61K007-06
CC 28-16 (Heterocyclic Compounds (More Than One Hetero Atom))
Section cross-reference(s): 1, **63**
OS CASREACT 117:90319; MARPAT 117:90319
AB Certain 5-fluoropyrimidine oxides and analogs thereof are claimed.

Their use for the manuf. of **pharmaceuticals** for the treatment of cardiovascular disorders and for the promotion **hair growth** is claimed. The use said 5-fluoropyrimidine compds. for the manuf. of **pharmaceuticals** contg. them admixed with minoxidil, vasoconstrictors, corticosteroids, triazine, scopolamine, **antiandrogens**, or 5-.alpha.-reductase **inhibitors** is claimed. Chlorination of 5-fluoro-4,6-dihydroxy-2-pyrimidinamine (phosphorous oxychloride/2-picoline) gave 4,6-dichloro-5-fluoro-2-pyrimidinamine (47% yield) which was aminated to give 6-chloro-5-fluoro-2,4-pyrimidinediamine (65% yield) and this was oxidized and aminated with piperidine to give 5-fluoro-6-(1-piperidinyl)-2,4-pyrimidinediamine 3-oxide (5-fluorominoxidil) (I). I **stimulated hair growth** in monkeys and I had antihypertensive activity.

ST fluorominoxidil **hair growth** antihypertensive; minoxidil fluoro **hair growth** antihypertensive

IT Antihypertensives
(fluorominoxidil)

IT Vasoconstrictors
(**hair growth** agents or antihypertensives contg. fluorominoxidil and)

IT Corticosteroids, biological studies
RL: RCT (Reactant)
(**hair growth** agents or antihypertensives contg. fluorominoxidil and)

IT **Androgens**
RL: RCT (Reactant)
(**antiandrogens, hair growth** agents or antihypertensives contg. fluorominoxidil and)

IT Cardiovascular system
(disease, treatment of, fluorominoxidil for)

IT **Hair** preparations
(**growth** stimulants, fluorominoxidil)

IT 110-89-4, Piperidine, reactions
RL: RCT (Reactant)
(amination with, of fluoropiperidinylpyrimidinediamine oxide)

IT 50-01-1, Guanidine hydrochloride
RL: RCT (Reactant)
(cyclocondensation reaction of, with di-Et fluoromalonate)

IT 685-88-1, Diethyl fluoromalonate
RL: RCT (Reactant)
(cyclocondensation reaction of, with guanidine hydrochloride)

IT 51-34-3, Scopolamine 290-87-9, s-Triazine 38304-91-5, Minoxidil
RL: RCT (Reactant)
(**hair growth** agents or antihypertensives contg. fluorominoxidil and)

IT 9036-43-5, 5.alpha.-Reductase
RL: USES (Uses)
(**inhibitors, hair growth** agents or antihypertensives contg. fluorominoxidil and)

IT 15598-33-1P, 4,6-Dichloro-5-fluoro-2-pyrimidinamine 142886-73-5P, 6-Chloro-5-fluoro-2,4-pyrimidinediamine 3-oxide
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation)
(prepn. and amination of)

IT 669-96-5P, 5-Fluoro-4,6-dihydroxy-2-pyrimidinamine
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation)
(prepn. and chlorination of)

IT 15047-12-8P, 6-Chloro-5-fluoro-2,4-pyrimidinediamine
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation)
(prepn. and oxidn. of)

IT 142886-74-6P, 5-Fluoro-6-(1-piperidinyl)-2,4-pyrimidinediamine 3-oxide
RL: SPN (Synthetic preparation); PREP (Preparation)

(prepn. of, as **hair growth** agent and antihypertensive)

L130 ANSWER 50 OF 97 WPIDS COPYRIGHT 1998 DERWENT INFORMATION LTD
AN 92-249834 [30] WPIDS
DNC C92-111451
TI **Reducing hair growth** by topical application of trans glutaminase **inhibitor** - esp. 3,5-di substd.-4,5-di hydro-isoxazole deriv., also making hair softer and easier to cut.
DC B03 D21
IN FUNKHOUSER, M G; HANDELMAN, J H; SHANDER, D
PA (HAND-I) HANDELMAN J H
CYC 3
PI WO 9211007 A1 920709 (9230)* EN 11 pp A61K031-42 <--
AU 9191653 A 920722 (9244) A61K031-42 <--
EP 563301 A1 931006 (9340) EN A61K031-42 <--
JP 06504057 W 940512 (9423) 5 pp A61K031-42 <--
AU 656550 B 950209 (9514) A61K031-42 <--
EP 563301 A4 931124 (9528) A61K031-42 <--
CA 2098102 C 961105 (9704) A61K031-42 <--
ADT WO 9211007 A1 WO 91-US9645 911219; AU 9191653 A AU 91-91653 911219, WO 91-US9645 911219; EP 563301 A1 WO 91-US9645 911219, EP 92-903695 911219; JP 06504057 W WO 91-US9645 911219, JP 92-503400 911219; AU 656550 B AU 91-91653 911219; EP 563301 A4 EP 92-903695 ; CA 2098102 C CA 91-2098102 911219
FDT AU 9191653 A Based on WO 9211007; EP 563301 A1 Based on WO 9211007; JP 06504057 W Based on WO 9211007; AU 656550 B Previous Publ. AU 9191653, Based on WO 9211007
PRAI US 90-632126 901220
REP 9.Jnl.Ref ; US 4720489; US 4912120; No-Citns.
IC ICM **A61K031-42**
ICS **A61K007-06; A61K007-15**
AB WO 9211007 A UPAB: 931006
The rate of mammalian **hair growth** is **reduced** and its character altered by applying to the skin of a mammal (not suffering from a disease characterised by elevated transglutaminase (TG) activity) a compsn. contg. a TG **inhibitor** (I).
(I) is pref. 5-(N-benzyloxycarbonyl) -1-phenylalanamidomethyl) -3-bromo-4,5-dihydroisoxazole (Ia), used at 10-2500 microg/sq.cm. of skin.
USE/ADVANTAGE - The method is esp. used to control **growth** of **androgen-stimulated hair**. Apart from **reducing growth**, (I) also makes the **hair** softer, downier and easier to cut. Topical compsns. contain 0.1-20% (I) plus usual carriers or vehicles.
0/0
FS CPI
FA AB; DCN
MC CPI: B07-E01; B12-L05; D08-B03

L130 ANSWER 51 OF 97 WPIDS COPYRIGHT 1998 DERWENT INFORMATION LTD
AN 92-041331 [05] WPIDS
TI Altering rate and characteristics of **hair growth** - by admin. of enzyme gamma glutamyl transpeptidase **inhibitor**.
DC B05
IN AHLUWALIA, G S; HARRINGTON, F E; SHANDER, D; AHLUWALIA, G
PA (HAND-I) HANDELMAN J H; (AHLU-I) AHLUWALIA G S
CYC 34
PI WO 9200069 A 920109 (9205)*
RW: AT BE DE DK ES FR GB GR IT LU NL OA SE

W: AT AU BB BG BR CA CH CS DE DK ES FI GB HU JP KP KR LK LU MC MG MW NL PL SD SE SU US

US 5096911 A 920317 (9214) 3 pp
AU 9182094 A 920227 (9218)
JP 06502389 W 940317 (9416) 4 pp A61K031-195 <--
EP 607124 A1 940727 (9429) EN A61K031-42 <--
R: AT BE CH DE DK ES FR GB GR IT LI LU NL SE
AU 663292 B 951005 (9547) A61K007-06 <--
EP 607124 B1 970813 (9737) EN 5 pp A61K031-42 <--
R: AT BE CH DE DK ES FR GB GR IT LI LU NL SE
DE 69127296 E 970918 (9743) A61K031-42 <--
ES 2104710 T3 971016 (9748) A61K031-42 <--

ADT US 5096911 A US 90-542586 900625; JP 06502389 W JP 91-511788 910621, WO 91-US4427 910621; EP 607124 A1 EP 91-912670 910621, WO 91-US4427 910621; AU 663292 B AU 91-82094 910621; EP 607124 B1 EP 91-912670 910621, WO 91-US4427 910621; DE 69127296 E DE 91-627296 910621, EP 91-912670 910621, WO 91-US4427 910621; ES 2104710 T3 EP 91-912670 910621

FDT JP 06502389 W Based on WO 9200069; EP 607124 A1 Based on WO 9200069; AU 663292 B Previous Publ. AU 9182094, Based on WO 9200069; EP 607124 B1 Based on WO 9200069; DE 69127296 E Based on EP 607124, Based on WO 9200069; ES 2104710 T3 Based on EP 607124

PRAI US 90-542586 900625

REP 3.Jnl.Ref ; US 4720489; 9.Jnl.Ref

IC **A61K031-34; A61K031-42**
ICM **A61K031-195; A61K031-42**
ICS **A61K031-34; A61K031-365**

ICA **A61K007-06**; C07D261-04

AB WO 9200069 A UPAB: 931006
Reducing the rate and altering the character of mammalian **hair growth** comprises applying to the skin a compsn. contg. an **inhibitor** of gamma-glyutaglytamyl transpeptidase (I).

The **inhibitor** is acivian, bromsulphalein or anthglutin applied at 10- 2500mg/cm2 skin. The **inhibitor** is incorporated in 0.1-20 wt.% non-toxic dermatologically acceptable vehicle.

USE/ADVANTAGE - Useful for altering the rate and character of mammalian **hair growth** pref. **androgen-stimulated hair growth**. @(10pp Dwg.No.0/0

FS CPI

FA AB; DCN

MC CPI: B06-A02; B07-E01; B10-A19; B12-G01B2; B12-L05

L130 ANSWER 52 OF 97 MEDLINE

AN 92256595 MEDLINE

DN 92256595

TI Effects of long-term anticonvulsant therapy on copper, zinc, and magnesium in hair and serum of epileptics.

AU Suzuki T; Koizumi J; Moroji T; Shiraishi H; Hori T; Baba A; Kawai N; Tada K

CS Department of Psychiatry, University of Tsukuba, Ibaraki, Japan.

SO BIOLOGICAL PSYCHIATRY, (1992 Mar 15) 31 (6) 571-81.
Journal code: A3S. ISSN: 0006-3223.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 199208

AB The effects of long-term anticonvulsant therapy on copper (Cu), zinc (Zn), and magnesium (Mg) in the serum and hair were investigated in epileptics. Hair concentrations of Cu in both male and female epileptics, Zn in male epileptics, and Mg in female epileptics were

significantly decreased when compared with those of age-matched and gender-matched controls. Hair Cu concentrations were significantly decreased in male epileptics; a significant decrease in hair Mg concentration was observed in female epileptics when compared with schizophrenics. An increased serum Cu concentration was found in female epileptics and a decreased Zn concentration was found in male epileptics. These findings suggest that long-term anticonvulsant therapy could induce alterations in both the metabolism and distribution of Cu, Zn, and Mg.

CT Check Tags: Female; Human; Male
Adult
Anticonvulsants: AD, administration & dosage
*Anticonvulsants: AE, adverse effects
Carbamazepine: AD, administration & dosage
Carbamazepine: AE, adverse effects
*Copper: BL, blood
Drug Therapy, Combination
Epilepsy, Generalized: BL, blood
*Epilepsy, Generalized: DT, drug therapy
***Hair: DE, drug effects**
Hair: ME, metabolism
Long-Term Care
*Magnesium: BL, blood
Middle Age
Phenobarbital: AD, administration & dosage
Phenobarbital: AE, adverse effects
Phenytoin: AD, administration & dosage
Phenytoin: AE, adverse effects
Schizophrenia: BL, blood
Valproic Acid: AD, administration & dosage
Valproic Acid: AE, adverse effects
*Zinc: BL, blood

RN 298-46-4 (Carbamazepine); **50-06-6 (Phenobarbital)**; 57-41-0 (Phenytoin); 7439-95-4 (Magnesium); 7440-50-8 (Copper); 7440-66-6 (Zinc); 99-66-1 (Valproic Acid)

CN 0 (Anticonvulsants)

L130 ANSWER 53 OF 97 BIOSIS COPYRIGHT 1998 BIOSIS

AN 93:302818 BIOSIS

DN BA96:21043

TI EFFECT OF NIZORAL ON THE FUNCTIONAL STATE OF THE HYPOTHALAMIC-PITUITARY-OVARIAN SYSTEM IN VIRULISM.

AU MANUSHAROVA R A

CS I.F. ZHORDAN RES. INST. HUMAN REPROD., MINIST. HEALTH GEORGIA, TBILISI, GEORGIA.

SO VRACH DELO 0 (8). 1992. 89-91. CODEN: VRDEA5 ISSN: 0049-6804

LA Russian

AB A study is presented of the effect of nisoral on the hypothalamo-pituitary-ovarian system in 25 patients with hyperandrogeny (ovarian in 11, suprarenal in 14). It was established that most patients with oligoamenorrhea and anovulation showed a restoration of the menstrual cycle after the 2-3 **treatment** courses and also absence of progression and **reduction** of the rate of pathological **hair growth**. After nisoral **treatment** the **testosterone** level decreased while estradiol and progesterone **increased**, gonadotropins remained unchanged, urinary excretion of 17-ketosteroids **reduced**.

ST HUMAN HORMONE-DRUG **TESTOSTERONE** ESTRADIOL PROGESTERONE GONADOTROPIN HYPERANDROGENY OLIGOMENORRHEA ANOVULATION PATHOLOGICAL **HAIR GROWTH**

RN 50-28-2 (ESTRADIOL)
57-83-0 (PROGESTERONE)
58-22-0 (TESTOSTERONE)

65277-42-1 (NIZORAL)
CC Circadian Rhythms and Other Periodic Cycles 07200
Biochemical Studies-General 10060
Biochemical Studies-Proteins, Peptides and Amino Acids 10064
Biochemical Studies-Sterols and Steroids 10067
Biochemical Studies-Carbohydrates 10068
Pathology, General and Miscellaneous-Therapy 12512
Reproductive System-Physiology and Biochemistry *16504
Reproductive System-Pathology *16506
Endocrine System-Gonads and Placenta *17006
Endocrine System-Pituitary *17014
Integumentary System-Pathology *18506
Nervous System-Physiology and Biochemistry *20504
Pharmacology-Clinical Pharmacology *22005
Pharmacology-Endocrine System *22016
Pharmacology-Reproductive System; Implantation Studies *22028
BC **Hominidae 86215**

L130 ANSWER 54 OF 97 HCAPLUS COPYRIGHT 1998 ACS
AN 1992:67222 HCAPLUS
DN 116:67222
TI Antidundruff and **hair-growth stimulating** **hair** tonics containing docosenoic acid or its derivatives
IN Katada, Tomonori; Kawaguchi, Shigetaka; Monobe, Akio; Fukunaga, Iwao; Kishi, Masataka
PA Nonogawa Shoji Y. K., Japan
SO Jpn. Kokai Tokkyo Koho, 8 pp.
CODEN: JKXXAF
PI JP 03206020 A2 910909 Heisei
AI JP 90-1712 900109
DT Patent
LA Japanese
IC ICM A61K007-06
ICA A61K007-075; A61K007-08; A61K007-11
CC 63-6 (**Pharmaceuticals**)
Section cross-reference(s): 1, 62
AB Hair tonics contain .gtoreq.1 compds. chosen from docosenoic acid and/or its derivs. as active ingredients, which prevent hair loss and scalop itching. A **hair** tonic was prepd. from 95% EtOH 94.0, erucic acid 4.0, and glycerin 2.0 wt. parts, which showed good **hair growth stimulating** effect in mice.
Erucic acid (0.5 mg) showed 100% **inhibition** of **testosterone** 5.alpha.-reductase.
ST hair tonic docosenoate deriv antidandruff; **hair growth stimulation** docosenoate deriv
IT Dandruff
(control of, hair tonics contg. docosenoic acid and/or its derivs. for)
IT Alopecia
(treatment of, hair tonics contg. docosenoic acid and/or its derivs. for)
IT Hair preparations
(tonics, contg. docosenoic acid and/or its derivs., antidandruff)
IT 112-86-7, Erucic acid 506-33-2, Brassidic acid 2752-99-0
25378-26-1, Docosenoic acid 25378-26-1D, Docosenoic acid, derivs., ammonium, alkali metal or alkaline earth salts 28063-42-5
28880-79-7 75626-91-4 81967-38-6 84083-00-1 102323-01-3
115785-27-8 138614-24-1
RL: BIOL (Biological study)
(hair tonics contg., antidandruff)

L130 ANSWER 55 OF 97 WPIDS COPYRIGHT 1998 DERWENT INFORMATION LTD
AN 91-361438 [49] WPIDS

CR 91-101460 [14]
DNC C91-155772
TI Nutritional supplement compsn. for hoof and coat - comprises methionine, biotin, yeast, solubles, chelated zinc and opt. carrier, preservative, antioxidant and flavour.
DC B05 C03 D13
IN MCCAULEY, C G
PA (MCCA-N) MCCAULEY BROTHERS I
CYC 1
PI US 5066498 A 911119 (9149)*
ADT US 5066498 A US 91-669673 910314
PRAI US 89-400830 890830; US 91-669673 910314
IC A23K001-00
AB US 5066498 A UPAB: 930928
A nutritional supplement compsn. for the hoof and coat of an animal comprises 0.0-96.0 palatable carrier, 2.0-50.0 methionine, 0.01-0.25 biotin, 2.0-20.0 live yeast culture and yeast fermentation solubles, 1.25-5.0 zinc in chelated form, 0.0-0.40 preservative, 0.0-1.5 antioxidant and 0.0-20.0 flavour. Figures are wt.%.

The carrier is pref. grain, esp. oatmeal feed, a flavour is cane molasses, preservatives are propionic acid, ammonium hydroxide, acetic acid, benzoic acid, sorbic acid and tartaric acid and their mixts. and an antioxidant is **ethoxyquin**. A pref. compsn. comprises 75.56 carrier, 10.5 DL-methionine, 0.07 biotin, 6.25 live yeast culture and yeast fermentation solubles, 5.0 flavour, 1.25 zinc methionine, 0.01 preservative and 0.02 antioxidant.

USE - The supplement provides a relatively inexpensive yet safe and effective means for the effective treatment of nutritional deficiencies adversely affecting the healthy **growth** of **hair** coat and hooves in domestic animals. A suitable feeding regimen is 0.5-3.0 oz. per day for at least 5 months.
0/0
FS CPI
FA AB; DCN
MC CPI: B04-B02B2; B05-A03A; B06-F03; B10-B02D; B12-L05; B12-L09; C04-B02B2; C05-A03A; C06-F03; C10-B02D; C12-L05; C12-L09; D03-G01

L130 ANSWER 56 OF 97 WPIDS COPYRIGHT 1998 DERWENT INFORMATION LTD
AN 91-101460 [14] WPIDS
DNC C91-043478
TI Treatment of hoof and coat ailments in animals - by admin. of a supplement comprising carrier, dl-methionine, yeast culture and zinc methionine etc..
DC B05 C03 D13
IN MCCAULEY, C G
PA (MCCA-N) MCCAULEY BROTHERS I
CYC 1
PI US 5000964 A 910319 (9114)*
ADT US 5000964 A US 89-400830 890830
PRAI US 89-400830 890830
IC A23K001-00
AB US 5000964 A UPAB: 930928
Hoof and coat ailments in animals resulting from nutritional deficiencies are treated by feeding the animals with 0.5-3 oz/day for at least 5 months of a compsn. comprising (by wt.) 0-95% palatable carrier, 2-50% DL-methionine (I),0.01-0.25% biotin (II), 2-20% live yeast culture and yeast fermentation solubles (III), 1.25% Zn methionine (IV), 0-0.4% preservative (V), 0-1.5% antioxidant (VI), and 0-20% flavouring agent (VII).

Carrier is pref. a grain, esp. oatmeal seed. Pref. (V) are EtCO2H, NH4OH, iOAc, PhCO2H, sorbic acid, tartaric acid or mixts.

Pref. (VI) contains **ethoxyquin**. Pref. (VII) is cane molasses.

ADVANTAGE - The compsns. are inexpensive to produce, yet provide efficient and effective treatment of nutritional deficiencies adversely affecting the healthy **growth** of **hair** coat and hooves of domestic animals.
0/0
FS CPI
FA AB; DCN
MC CPI: B04-A07D2; B04-B02B2; B04-D01; B05-A03A; B05-C01; B06-D02; B06-F03; B10-B02D; B10-C02; B10-C04C; B10-C04E; B12-A07; B12-L09; C04-A07D2; C04-B02B2; C04-D01; C05-A03A; C05-C01; C06-D02; C06-F03; C10-B02D; C10-C02; C10-C04C; C10-C04E; C12-A07; C12-L09; D03-G01

L130 ANSWER 57 OF 97 HCAPLUS COPYRIGHT 1998 ACS
AN 1991:136036 HCAPLUS
DN 114:136036
TI Localization of minoxidil **sulfotransferase** in rat liver and the outer root sheath of anagen pelage and vibrissa follicles
AU Dooley, Thomas P.; Walker, Cynthia J.; Hirshey, Sharon J.; Falany, Charles N.; Diani, Arthur R.
CS Upjohn Co., Kalamazoo, MI, 49001, USA
SO J. Invest. Dermatol. (1991), 96(1), 65-70
CODEN: JIDEAE; ISSN: 0022-202X
DT Journal
LA English
CC 1-12 (Pharmacology)
AB The precise biochem. mechanism and site(s) of action by which minoxidil stimulates **hair growth** are not yet clear. Minoxidil sulfate is the active metabolite of minoxidil, with regard to smooth muscle vasodilation and **hair growth**. Formation of minoxidil sulfate is catalyzed by specific PAPS-dependent **sulfotransferase**(s) and minoxidil-sulfating activities have been previously reported to be present in liver and **hair** follicles. One of these minoxidil-sulfating enzymes has been purified from rat liver (rat minoxidil **sulfotransferase**, MST) and a rabbit anti-MST antibody has been prepd. Using this anti-MST antibody, the authors have immunohistochem. localized minoxidil **sulfotransferase** in the liver and anagen **hair** follicles from the rat. In rat prelage and vibrissa follicles, this enzyme is localized within the cytoplasm of epithelial cells in the lower outer root sheath. Although the immunolocalization of MST might not necessarily correlate with the MST activity known to be present in anagen follicles, the results of this study strongly suggest that the lower outer root sheath of the **hair** follicle may serve as a site for the sulfation of topically applied minoxidil.
ST minoxidil **sulfotransferase** liver **hair** follicle
IT **Hair**
(**growth** of, minoxidil stimulation of, minoxidil **sulfotransferase** of follicle in)
IT **Hair**
(follicle, minoxidil **sulfotransferase** of, **hair** -**growth** stimulation in relation to)
IT 83701-22-8, Minoxidil sulfate
RL: BIOL (Biological study)
(as **hair growth**-stimulating metabolite of minoxidil, minoxidil **sulfotransferase** of **hair** follicle in relation to)
IT 38304-91-5, Minoxidil
RL: BIOL (Biological study)
(**hair-growth** stimulation by, minoxidil **sulfotransferase** of **hair** follicle in relation to)
IT 129924-25-0, Minoxidil **sulfotransferase**

RL: BIOL (Biological study)
(of **hair** follicle, **hair-growth** stimulation by minoxidil in relation to)

L130 ANSWER 58 OF 97 WPIDS COPYRIGHT 1998 DERWENT INFORMATION LTD
AN 90-321544 [43] WPIDS
DNC C90-139220
TI Compsn. for external application contg. 2-di methylamino-ethanol - to improve skin condition, **reduce** hair loss, etc..
DC B05 D21 E16
PA (ASCH-N) ASCHEMIE MULLER R; (MUEL-N) MUELLER AZCHEMIE ROBERT; (MULL-I) MULLER R
CYC 14
PI DE 3912477 A 901018 (9043)*
EP 396857 A 901114 (9046)
R: AT BE CH DE ES FR GB GR IT LI LU NL SE
JP 02292215 A 901203 (9103)
JP 06018775 B2 940316 (9414) 3 pp A61K031-13 <--
ADT DE 3912477 A DE 89-3912477 890415; EP 396857 A EP 90-102853 900214; JP 02292215 A JP 90-96166 900411; JP 06018775 B2 JP 90-96166 900411
FDT JP 06018775 B2 Based on JP 02292215
PRAI DE 89-3912477 890415
REP 1.Jnl.Ref ; DE 2131946; GB 1182320
IC **A61K007-48; A61K031-13**
ICM **A61K031-13**
ICS **A61K007-06; A61K007-48**
AB DE 3912477 A UPAB: 940421
Compsn. for external application contains 2-dimethylaminoethanol (I) plus usual formulation materials. (I) is used as a salt or ester, esp. the hydrogencarbonate, citrate, orotate, hydrogentartrate, aceglutamate, acetamidobenzoate or hydrogensuccinate.
USE/ADVANTAGE - The compsns. improve the condition (elasticity and structure) of the skin, preventing premature ageing and development of wrinkles. They also **reduce** **androgen**-dependent **hair** loss and **stimulate** **hair growth**. (I) **increases** protein synthesis and prolongs the livetime of (post)mitotic fibroblasts. (I) is already known for internal use as a psychopharmaceutical and for treatment of geriatric disorders. @(3pp Dwg.No.0/0)
0/0
FS CPI
FA AB; DCN
MC CPI: B10-B03B; B12-A07; B12-L02; B12-L05; D08-B03; D08-B09A; E10-B03B

L130 ANSWER 59 OF 97 HCAPLUS COPYRIGHT 1998 ACS
AN 1991:628473 HCAPLUS
DN 115:228473
TI Are phytohormones involved in plant-rhizobium interaction?
AU Prisen, E.; Chauvaux, N.; Schmidt, J.; John, M.; De Greef, J.; Van Onckelen, H.
CS Dep. Biol., Univ. Antwerp, Wilrijk, B-2610, Belg.
SO Meded. Fac. Landbouwwet., Rijksuniv. Gent (1990), 55(4), 1393-401
CODEN: MFLRA3; ISSN: 0368-9697
DT Journal
LA English
CC 11-3 (Plant Biochemistry)
AB Initial stages of Rhizobium-plant interaction include root **hair** deformation (had) and root **hair** curling (hac). These stages are correlated with **growth** changes in plant epidermal root hairs and initiation of cell division in the cortex of the host root. Recently, this had and/or hac factor is shown to be a lipooligosaccharide and therefore unlike any of the main endogenous plant hormone types. Although IAA is present in

Rhizobium culture filtrates the correlation between the ability to nodulate and the ability to produce IAA is still unclear. To investigate the possible role of IAA in the early stage of Rhizobium-plant interaction, IAA synthesis was studied in Rhizobium cultures in presence or absence of flavonoids. Only addn. of luteolin and naringenin to R. meliloti or R. leguminosarum culture resulted in increased IAA-levels from the early stationary phase on. This relates to the strain-specific nod-gen induction by these flavonoids. Once inoculated the endogenous IAA levels in the roots remained unchanged.

ST IAA plant Rhizobium interaction

IT Flavonoids
RL: BIOL (Biological study)
(IAA formation by Rhizobium in presence of)

IT Rhizobium leguminosarum
Rhizobium meliloti
(plant interaction with, IAA formation during)

IT Alfalfa
(Rhizobium interaction with, IAA formation in relation to)

IT Symbiosis
(alfalfa-Rhizobium, hormones in)

IT **480-41-1**, Naringenin 491-70-3, Luteolin
RL: BIOL (Biological study)
(IAA formation by Rhizobium in presence of)

IT 87-51-4, IAA, biological studies
RL: FORM (Formation, nonpreparative)
(formation of, during plant-Rhizobium interactions)

L130 ANSWER 60 OF 97 HCAPLUS COPYRIGHT 1998 ACS

AN 1990:584178 HCAPLUS

DN 113:184178

TI Sulfation of minoxidil by human liver phenol **sulfotransferase**

AU Falany, Charles N.; Kerl, Elizabeth A.

CS Cancer Cent., Univ. Rochester, Rochester, NY, 14642, USA

SO Biochem. Pharmacol. (1990), 40(5), 1027-32
CODEN: BCPCA6; ISSN: 0006-2952

DT Journal

LA English

CC 1-2 (Pharmacology)
Section cross-reference(s): 7

AB The N-Q-sulfate of minoxidil (I) is the active agent in producing the vasodilation and the **hair-growth** stimulating responses obsd. with I treatment. In this report, I sulfation activity was assayed in cytosol prepd. from several normal human livers, and I sulfation was shown to correlate with the activity of the phenol-sulfating form of phenol **sulfotransferase** (P-PST) activity in the same livers. No correlation was obsd. between I sulfation and the dopamine or dehydroepiandrosterone (DHEA) **sulfotransferase** activities present in human liver. I sulfation also copurified with P-PST activity during the purifn. of P-PST from human liver. During the purifn. procedure, I and p-nitrophenol **sulfotransferase** (P-PST) activities were resolved from the dopamine and DHEA sulfation activities catalyzed by the monoamine-sulfating form of phenol **sulfotransferase** (M-PST) and DHEA **sulfotransferase** resp. Also, purified DHEA **sulfotransferase** was not capable of sulfating I, and no data were obtained to indicate that I is a substrate for M-PST. p-Nitrophenol, a substrate for P-PST, was demonstrated to be a competitive inhibitor of I sulfation catalyzed by purified P-PST when I was the variable substrate. These results indicate that I is sulfated and, therefore, bioactivated by P-PST in human liver.

ST minoxidil sulfation liver phenol **sulfotransferase**

IT Liver, metabolism

(minoxidil sulfation in human)
IT Sulfation
(of minoxidil, in human liver)
IT 9026-08-8
RL: PRP (Properties)
(activity of, in human liver)
IT 9023-09-0, **Sulfotransferase** 9026-09-9, Phenol **sulfotransferase**
RL: PRP (Properties)
(activity of, in human liver, minoxidil metab. in relation to)
IT 83701-22-8
RL: FORM (Formation, nonpreparative)
(formation of, in human liver)
IT 38304-91-5, Minoxidil
RL: RCT (Reactant)
(sulfation of, in human liver, by phenol **sulfotransferase**)

L130 ANSWER 61 OF 97 BIOSIS COPYRIGHT 1998 BIOSIS DUPLICATE 7
AN 90:358055 BIOSIS
DN BA90:54634
TI RECONSTITUTED EPIDERMIS A NOVEL MODEL FOR THE STUDY OF DRUG METABOLISM IN HUMAN EPIDERMIS.
AU PHAM M-A; MAGDALOU J; SIEST G; LENOIR M-C; BERNARD B A; JAMOULLE J-C; SHROOT B
CS CENT. INTERNATIONAL DE RECHERCHES DERMATOLOGIQUES, SOPHIA ANTIPOLIS, F-06565 VALBONNE, FRANCE.
SO J INVEST DERMATOL 94 (6). 1990. 749-752. CODEN: JIDEAE ISSN: 0022-202X
LA English
AB The metabolic capacity of reconstituted epidermis from the outer root sheath cell of human **hair** follicles was determined. It was found that this epidermis possesses enzymes involved in both phase I (oxidation) and phase II (conjugation) reactions for drug biotransformation. The use of model substrates allowed the characterization of several isoenzymes. The homogenate fraction contained membrane-bound mixed-function oxidases (cytochrome P-450 dependent) involved in the O-dealkylation of 7-ethoxy-, and 7-benzoxyresorufin, NADPH cytochrome c (P-450) reductase, testosterone 5.alpha.-reductase, and UDP-**glucuronosyltransferases**, which conjugate 1-naphthol and bilirubin. One isoform of each glutathione S-transferase, steroid-, and arylsulfatases, acting on estrone- and 4-methylumbelliforme sulfates, were detected. Additionally, the activity of two distinct forms of epoxide hydrolases, which hydrate cis- and trans-stilbene oxides, could be measured. The presence of these drug metabolizing enzymes in the reconstituted epidermis indicates that it has a potential to serve as a model to study epidermal drug metabolism in vitro.
ST NADPH CYTOCHROME C REDUCTASE TESTOSTERONE 5-ALPHA-REDUCTASE GLUTATHIONE S-TRANSFERASE MIXED-FUNCTION OXIDASE EPOXIDE HYDROLASE ARYLSULFATASE STEROID SULFATASE UDP-**GLUCURONOSYLTRANSFERASE** PHARMACOKINETICS BIOTRANSFORMATION
RN 58-22-0 (TESTOSTERONE)
9016-17-5 (ARYLSULFATASE)
9025-62-1 (STEROID SULFATASE)
9030-08-4 (UDP-GLUCURONOSYLTRANSFERASE)
9035-73-8 (OXIDASE)
9048-63-9 (EPOXIDE HYDROLASE)
50812-37-8 (GLUTATHIONE S-TRANSFERASE)
9023-03-4Q, 78519-49-0Q (NADPH CYTOCHROME C REDUCTASE)
CC Biochemical Studies-General 10060
Biochemical Studies-Proteins, Peptides and Amino Acids 10064
Biochemical Studies-Porphyrins and Bile Pigments 10065

Biochemical Studies-Sterols and Steroids 10067
Enzymes-Physiological Studies *10808
Metabolism-General Metabolism; Metabolic Pathways *13002
Metabolism-Sterols and Steroids 13008
Metabolism-Proteins, Peptides and Amino Acids *13012
Metabolism-Porphyrins and Bile Pigments 13013
Blood, Blood-Forming Organs and Body Fluids-Blood and Lymph Studies *15002
Integumentary System-Physiology and Biochemistry *18504
Pharmacology-Drug Metabolism; Metabolic Stimulators *22003
Pharmacology-Integumentary System, Dental and Oral Biology *22020
Routes of Immunization, Infection and Therapy 22100
BC Hominidae 86215

L130 ANSWER 62 OF 97 HCAPLUS COPYRIGHT 1998 ACS
AN 1990:567833 HCAPLUS
DN 113:167833
TI Purification and characterization of rat liver minoxidil **sulfotransferase**
AU Hirshey, Sharon J.; Falany, Charles N.
CS Cancer Cent., Univ. Rochester, Rochester, NY, 14642, USA
SO Biochem. J. (1990), 270(3), 721-8
CODEN: BIJOAK; ISSN: 0306-3275
DT Journal
LA English
CC 7-2 (Enzymes)
AB Minoxidil (Mx), a pyrimidine N-oxide, is used therapeutically as an antihypertensive agent and to induce **hair growth** in patients with male pattern baldness. Mx NO-sulfate has been implicated as the agent active in producing these effects. This paper describes the purifn. of a unique **sulfotransferase** (ST) from rat liver cytosol that is capable of catalyzing the sulfation of Mx. By using DEAE-Sepharose CL-6B chromatog., hydroxylapatite chromatog. and ATP-agarose affinity chromatog., Mx-ST activity was purified 240-fold compared with the activity in cytosol. The purified enzyme was also capable of sulfating p-nitrophenol (PNP) at low concns. (less than 10 .mu.M). Mx-ST was purified to homogeneity, as evaluated by SDS/PAGE and reverse-phase HPLC. The active form of the enzyme had a mol. mass of 66,000-68,000 Da as estd. by gel exclusion chromatog. and a subunit mol. mass of 35,000 Da. The apparent Km values for Mx, 3'-phosphoadenosine 5'-phosphosulfate, and PNP were 625, 5.0, and 0.5 .mu.m, resp. However, PNP displayed potent substrate inhibition at concns. above 1.2 .mu.M. Antibodies raised in rabbits to the pure enzyme detected a single band in rat liver cytosol with a subunit mol. mass of 35,000 Da, as detd. by immunoblotting. The anti-(rat Mx-ST) antibodies also reacted with the phenol-sulfating form of human liver phenol **sulfotransferase**, suggesting some structural similarity between these proteins.
ST minoxidil **sulfotransferase** liver
IT Liver, composition
(minoxidil **sulfotransferase** of, purifn. and characterization of)
IT Michaelis constant
(of minoxidil **sulfotransferase**, of liver)
IT Amino acids, biological studies
RL: BIOL (Biological study)
(of minoxidil **sulfotransferase**, of liver)
IT 129924-25-0P
RL: PREP (Preparation)
(of liver cytosol, purifn. and characterization of)
IT 9026-09-9
RL: PROC (Process)
(of liver, isolation of, minoxidil **sulfotransferase** in

relation to)
IT 100-02-7, reactions 482-67-7, 3'-Phosphoadenosine 5'-phosphosulfate 38304-91-5, Minoxidil
RL: RCT (Reactant)
(reaction of, with minoxidil **sulfotransferase** of liver, kinetics of)

L130 ANSWER 63 OF 97 HCAPLUS COPYRIGHT 1998 ACS
AN 1991:157150 HCAPLUS
DN 114:157150
TI Minoxidil sulfate is the active metabolite that stimulates **hair** follicles
AU Buhl, Allen E.; Waldon, Daniel J.; Baker, Carolyn A.; Johnson, Garland A.
CS Hairgrowth Res., Upjohn Co., Kalamazoo, MI, USA
SO J. Invest. Dermatol. (1990), 95(5), 553-7
CODEN: JIDEAE; ISSN: 0022-202X
DT Journal
LA English
CC 1-12 (Pharmacology)
AB An important step in understanding minoxidil's mechanism of action on **hair** follicles was to det. the drug's active form. Organ-cultured vibrissa follicles were used to test whether it is minoxidil or its sulfated metabolite, minoxidil sulfate, that stimulates **hair growth**. Follicles from neonatal mice were cultured with or without drugs and effects were assessed by measuring incorporation of radiolabeled cysteine in **hair** shafts of the treated follicles. Assays of minoxidil **sulfotransferase** activity indicated that vibrissae follicles metabolize minoxidil to minoxidil sulfate. Dose-response studies showed that minoxidil sulfate is 14 times more potent than minoxidil in stimulating cysteine incorporation in cultured follicles. Three drugs that block prodn. of intrafollicular minoxidil sulfate were tested for their effects on drug-induced **hair growth**. Diethylcarbamazine proved to be a noncompetitive inhibitor of **sulfotransferase** and prevented **hair growth** stimulation by minoxidil but not by minoxidil sulfate. Inhibiting the formation of intracellular PAPS with chlorate also blocked the action of minioxidil but not of minoxidil sulfate. Acetaminophen, a potent sulfate scavenger, blocked cysteine incorporation by minoxidil. It also blocked follicular stimulation by minoxidil sulfate apparently by directly removing the sulfate from the drug. Expts. with U-51,607, a potent minoxidil analog that also forms a sulfated metabolite, showed that its activity was inhibited by both chlorate and diethylcarbamazine. These studies show that sulfation is a crit. step for **hair -growth** effects of minoxidil and that it is the sulfated metabolite that directly affects **hair** follicles.
ST minoxidil sulfate **hair growth**
IT Drug interactions
(of acetaminophen with minoxidil sulfation, **hair** follicle stimulation in relation to)
IT **Hair**
(follicle, stimulation of, by minoxidil sulfate)
IT 103-90-2
RL: BIOL (Biological study)
(**hair** follicle stimulation by minoxidil inhibition by)
IT 38304-91-5, Minoxidil 132971-00-7, U 51607
RL: BIOL (Biological study)
(**hair** follicle stimulation by, sulfate metabolite in)
IT 83701-22-8
RL: BIOL (Biological study)
(**hair** follicles stimulation by, as minoxidil metabolite)

IT 52-90-4, Cysteine, biological studies
RL: BIOL (Biological study)
(in **hair** follicle stimulation by minoxidil, sulfate metabolite formation in relation to)

L130 ANSWER 64 OF 97 HCAPLUS COPYRIGHT 1998 ACS
AN 1990:492274 HCAPLUS
DN 113:92274
TI The ENOD12 gene product is involved in the infection process during the pea-Rhizobium interaction
AU Scheres, Ben; Van de Wiel, Clemens; Zalensky, Andrei; Horvath, Beatrix; Spaink, Herman; Van Eck, Herman; Zwartkruis, Fried; Wolters, Anne Marie; Gloudemans, Ton; et al.
CS Dep. Mol. Biol., Agric. Univ., Wageningen, 6703 HA, Neth.
SO Cell (Cambridge, Mass.) (1990), 60(2), 281-94
CODEN: CELLB5; ISSN: 0092-8674
DT Journal
LA English
CC 3-3 (Biochemical Genetics)
Section cross-reference(s): 6, 11
AB The pea cDNA clone pPsENOD12 represents a gene involved in the infection process during Pisum sativum-Rhizobium leguminosarum bv. viciae symbiosis. The ENOD12 protein is composed of pentapeptides contg. 2 hydroxyprolines. The expression of the ENOD12 gene is induced in cells through which the infection thread is migrating, but also in cells that do not yet contain an infection thread. Sol. compds. from Rhizobium are involved in eliciting ENOD12 gene expression. Rhizobium common and host-specific nodulation genes are essential for the prodn. of these compds. Two ENOD12 genes are expressed in nodules and in stem tissue of uninoculated plants. The gene represented by the cloned ENOD12 mRNA is also expressed in flowers, but a different transcription start may be used.
ST pea nodulin ENOD12 cDNA sequence; nodulin ENOD12 gene pea Rhizobium infection; flower stem pea nodulin ENOD12 gene
IT Gene and Genetic element, plant
(for nodulin ENOD12 of pea, involved in Rhizobium leguminosarum infection process, sequence and expression and regulation of)
IT Rhizobium leguminosarum viceae
(genes nod and excreted compds. of, nodulin ENOD12 gene expression requirement for, in pea interaction)
IT Flower
Stem
(nodulin ENOD12 gene expression in, of pea)
IT Root nodule
(nodulin ENOD12 gene expression in, of pea during Rhizobium leguminosarum interaction)
IT Pea
(nodulin ENOD12 of, involved in Rhizobium leguminosarum infection process, sequence and expression and regulation of)
IT Plant **growth** and development
(of nodules, in pea-Rhizobium leguminosarum interaction, nodulin ENOD12 gene expression during)
IT Protein sequences
(of nodulin ENOD12 and precursor of pea, complete)
IT Root
(cortex, nodulin ENOD12 gene expression in, of pea, during Rhizobium leguminosarum infection process)
IT Root
(**hair**, nodulin ENOD12 gene expression in, of pea, during Rhizobium leguminosarum infection process)
IT Proteins, specific or class
RL: BIOL (Biological study)
(hydroxyproline-rich, nodulin ENOD12 of pea as)
IT Deoxyribonucleic acid sequences

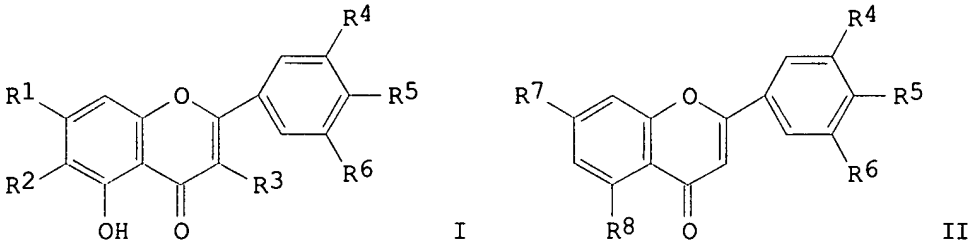
(nodulin ENOD12-specifying, of pea, complete)
IT Proteins, specific or class
RL: BIOL (Biological study)
(nodulins ENOD12 (early nodulin 12), of pea, involved in Rhizobium leguminosarum infection process, sequence and expression and regulation of)
IT Symbiosis
(pea-Rhizobium leguminosarum, nodulin ENOD12 gene expression and regulation in)
IT Gene and Genetic element, microbial
RL: BIOL (Biological study)
(nod, of Rhizobium leguminosarum viciae, nodulin ENOD12 gene expression requirement for, in pea interaction)
IT 128768-95-6, Nodulin 12 (pea clone pPsENOD12) 128770-79-6, Nodulin 12 (pea clone pPsENOD12 precursor)
RL: PRP (Properties)
(amino acid sequence of)
IT **480-41-1**, Naringenin
RL: PRP (Properties)
(nodulin ENOD12 gene of pea activation by Rhizobium leguminosarum viciae **grown** in)
IT 128769-96-0, Deoxyribonucleic acid (pea clone pPsENOD12 nodulin 12 messenger RNA-complementary)
RL: BIOL (Biological study); PRP (Properties)
(nucleotide sequence of)

L130 ANSWER 65 OF 97 HCAPLUS COPYRIGHT 1998 ACS
AN 1990:104597 HCAPLUS
DN 112:104597
TI Cosmetics containing unsaturated fatty acids, antioxidants, amino acids, and polybasic acids
IN Kato, Hisatoyo; Shimizu, Mitsuaki; Ozasa, Yoshiji
PA Sunstar, Inc., Japan
SO Jpn. Kokai Tokkyo Koho, 6 pp.
CODEN: JKXXAF
PI JP 01216908 A2 890830 Heisei
AI JP 88-42837 880224
DT Patent
LA Japanese
IC ICM A61K007-00
ICS A61K007-06
CC 62-4 (Essential Oils and Cosmetics)
AB Cosmetics contain (a) .gtoreq.2 unsatd. bonds-contg. C18-22 fatty acids, their salts, or their esters with mono- or di-hydric alcs., (b) .gtoreq.1 antioxidants chosen from dibutylhydroxytoluene, butylhydroxyanisole, erythorbic acid, Na erythorbate, nordihydroguaiaretic acid, Pr gallate, a sage ext., a rosemary ext., and a mace ext., (c) amino acids and/or their esters, and (d) .gtoreq.1 compds. chosen from aliph. hydroxy polybasic acids, their salts, their mono esters, and carboxyvinyl polymers. The C18-22 fatty acids, which show moisture-retaining, tyrosinase-inhibiting, and **hair growth** stimulating effects, are stabilized in the cosmetics. A lotion comprised linoleic acid 0.5, dibutylhydroxytoluene 0.05, citric acid 0.05, poly(oxyethylene) hydrogenated castor oil 1.0, Me p-hydroxybenzoate 0.05, EtOH 15.0, glycerin 8.0, KOH 0.15, Na tartrate 0.03, glycine 0.05, L-serine 0.05, L-cystine 0.001, fragrance 0.1, and H2O to 100 wt.%.
ST fatty ester cosmetic
IT Cosmetics
(contg. unsatd. fatty acids and antioxidants and amino acids and polybasic acids, with stability)
IT Antioxidants
(cosmetics contg. unsatd. fatty acids and amino acids and polybasic acids and, for stability)

IT Amino acids, biological studies
RL: BIOL (Biological study)
(cosmetics contg. unsatd. fatty acids and antioxidants and polybasic acids and, for stability)
IT Mace (spice)
Rosemary
Sage
(exts., cosmetics contg. unsatd. fatty acids and amino acids and polybasic acids and, for stability)
IT Fatty acids, biological studies
RL: BIOL (Biological study)
(C18-22-unsatd., cosmetics contg. antioxidants and amino acids and polybasic acids and, for stability)
IT Vinyl compounds, polymers
RL: BIOL (Biological study)
(carboxy-contg., polymers, cosmetics contg. unsatd. fatty acids and antioxidants and amino acids and, for stability)
IT 60-33-3, Linoleic acid, biological studies 463-40-1, .alpha.-Linolenic acid 506-21-8 506-26-3, .gamma.-Linolenic acid 544-35-4, Ethyl linoleate 1808-26-0, Ethyl arachidonate 22882-95-7, Isopropyl linoleate .
RL: BIOL (Biological study)
(cosmetics contg. antioxidants and amino acids and polybasic acids and, for stability)
IT 89-65-6, Erythorbic acid 121-79-9, Propyl gallate 500-38-9, Nordihydroguaiaretic acid 6381-77-7, Sodium erythorbate **25013-16-5**, Butylhydroxyanisole **30587-81-6**, Dibutylhydroxytoluene
RL: BIOL (Biological study)
(cosmetics contg. unsatd. fatty acids and amino acids and polybasic acids and, for stability)
IT 68-04-2, Sodium citrate 77-92-9, biological studies 87-69-4, biological studies 526-95-4, Gluconic acid 6915-15-7, Malic acid 14475-11-7 39413-05-3, Isopropyl citrate
RL: BIOL (Biological study)
(cosmetics contg. unsatd. fatty acids and antioxidants and amino acids and, for stability)
IT 56-40-6, Glycine, biological studies 56-45-1, L-Serine, biological studies 56-84-8, L-Aspartic acid, biological studies 56-85-9, L-Glutamine, biological studies 56-86-0, L-Glutamic acid, biological studies 56-89-3, L-Cystine, biological studies 59-51-8, DL-Methionine 60-18-4, L-Tyrosine, biological studies 61-90-5, L-Leucine, biological studies 70-47-3, L-Asparagine, biological studies 71-00-1, L-Histidine, biological studies 72-18-4, L-Valine, biological studies 72-19-5, L-Threonine, biological studies 73-22-3, L-Tryptophan, biological studies 73-32-5, L-Isoleucine, biological studies 74-79-3, L-Arginine, biological studies 80-68-2, DL-Threonine 147-85-3, L-Proline, biological studies 150-30-1, DL-Phenylalanine 338-69-2, D-Alanine 4070-48-8, L-Valine methyl ester 7555-06-8, L-Histidine ethyl ester 10098-89-2, L-Lysine hydrochloride 13827-65-1, Glycine lauryl ester
RL: BIOL (Biological study)
(cosmetics contg. unsatd. fatty acids and antioxidants and polybasic acids and, for stability)

L130 ANSWER 66 OF 97 HCAPLUS COPYRIGHT 1998 ACS
AN 1989:601382 HCAPLUS
DN 111:201382
TI 5.alpha.-Reductase inhibiting agnets containing flavonoids
IN Okuda, Minehiro; Kawai, Michio; Imokawa, Genji; Akatsu, Mitsuhiro; Takaishi, Naotake
PA Kao Corp., Japan
SO Jpn. Kokai Tokkyo Koho, 7 pp.

CODEN: JKXXAF
PI JP 01096126 A2 890414 Heisei
AI JP 87-254250 871008
DT Patent
LA Japanese
IC ICM A61K031-35
ICS A61K031-70
CC 62-3 (Essential Oils and Cosmetics)
Section cross-reference(s): 1, 7, 63
OS MARPAT 111:201382
GI



AB 5.alpha.-Reductase inhibiting agents, useful as drugs and **hair growth** stimulants, contain flavonoids I and II (R1 = H, OH, glucuronic acid residue; R2, R4, R5-8 = H, OH; R3 = H, OH, sugar residue) as active ingredients. Baicalin inhibited 90.3% 5.alpha.-reductase in vitro, vs. 82.3%, for oxendolone. Aq. EtOH soln. contg. 3 wt.% baicalein was applied to male patients with **alopecia** for 2 mo to show **hair growth**.
ST flavonoid reductase inhibitor **alopecia**
IT Flavonoids
RL: BIOL (Biological study)
(5.alpha.-reductase inhibitors contg., for **hair growth** enhnacement)
IT **Alopecia**
(treatment of, 5.alpha.-reductase inhibiting flavonoids for)
IT **Hair** preparations
(**growth** stimulants, contg. 5.alpha.-reductase inhibiting flavonoids)
IT 117-39-5, Quercetin 153-18-4, Rutin **486-66-8** 491-67-8, Baicalein 520-18-3, Kaempferol 21967-41-9, Baicalin
RL: BIOL (Biological study)
(5.alpha.-reductase inhibitors contg., for **hair growth** enhancement)
IT 9081-34-9
RL: BIOL (Biological study)
(inhibitors for, flavonoids as, for **hair growth** enhnacement)

L130 ANSWER 67 OF 97 WPIDS COPYRIGHT 1998 DERWENT INFORMATION LTD
AN 90-044777 [06] WPIDS
CR 85-159178 [26]
DNC C90-019539
TI Alteration of character of male beard growth - by topical administration of 5-alpha-**reductase inhibitor** and/or cytoplasmic **androgen** receptor binding agent.
DC B01
IN KASZYNSKI, E G; SHANDER, D; USDIN, V R; VANDERLEE, H
PA (BREU-I) BREUER M M
CYC 1
PI US 4885289 A 891205 (9006)* 7 pp
KATHLEEN FULLER BT/LIBRARY 308-4290

ADT US 4885289 A US 85-807623 851211
PRAI US 83-560726 831212; US 85-807623 851211
IC **A61K031-56**
AB US 4885289 A UPAB: 950602
A process for **reducing** the rate and altering the character toward the vellus state of **androgen-stimulated** beard **hair growth** in intact, sexually mature males comprises applying to the skin a compsn. contg. a 5-alpha-**reductase inhibitor** (I) and/or a cytoplasmic **androgen** receptor binding agent (II).
(I) may be e.g. progesterone, (4R)-5,10-seco-19-norpregna 4,5-diene-3,10,20-trione or 4-androstene-3-one 17beta-carboxylic acid. (II) may be e.g. cyproterone acetate, chlormadinone acetate, 17alpha-**propyltestosterone** or spironolactone. Also claimed is a process for **reducing** the forces required to cut **androgen-stimulated** beard hair in intact sexually mature males which comprises applying to the skin a compsn. contg. (I) and/or (II).
USE/ADVANTAGE - The normal rate of male beard growth can be **reduced** and its character caused to revert toward the vellus state, with accompanyingr **redn.** in cutting force by the topical administration of (I) or (II). Unwanted interference with other **androgen** mediated bodily processes can be minimized or avoided.
0/0
Dwg.0/0
FS CPI
FA AB; DCN
MC CPI: B01-B04; B01-C03; B01-C04; B01-C05; B01-C10; B01-D01; B01-D02; B12-A07; B12-G01B1; B12-G04A; B12-K04A; B12-L05

L130 ANSWER 68 OF 97 BIOSIS COPYRIGHT 1998 BIOSIS
AN 89:476257 BIOSIS
DN BA88:112017
TI ANIMAL MODELS OF **ANDROGEN**-DEPENDENT DISORDERS OF THE PILOSEBACEOUS APPARATUS 1. THE ANDROCHRONOGENETIC ALOPECIA AGA MOUSE AS A MODEL FOR MALE-PATTERN BALDNESS.
AU MATIAS J R; MALLOY V; ORENTREICH N
CS ORENTRICH FOUNDATION ADVANCEMENT SCI. INC., BIOMED. RES. STATION, RD 2 BOX 375, COLD SPRING-ON-HUDSON, N.Y. 10516.
SO ARCH DERMATOL RES 281 (4). 1989. 247-253. CODEN: ADREDL ISSN: 0340-3696
LA English
AB The androchronogenetic alopecia (AGA) mouse is a mutant strain which expresses **androgen**-dependent baldness. Daily s.c. injection of **testosterone** (T) induced thinning of the **hair** coat along the upper dorsum after 4 weeks of **treatment**. After 12 to 14 weeks this diffuse alopecia eventually developed into a bald area which extended to the middorsum. Dihydrotestosterone was more effective than T in **stimulating** the onset of AGA. In this model, T produced the alopecia by decreasing the rate of **hair growth**, decreasing the duration of anagen, and markedly prolonging the duration of teolgen. When applied topically at a concentration of 5%, cyproterone acetate delayed the progression of the T-mediated **hair** loss. However, this **inhibitory** effect occurred through systemic means as evidenced by decrease in the size of the submaxillary gland. Chronic feeding of **androgen-treated** female AGA mice with a diet containing 0.01% minoxidil also **inhibited** the development of alopecia. Skin and core temperatures were found to be higher in minoxidil-**treated** animals than in the placebo-**treated** controls. Minoxidil at a topical dose of 1% did not produce any effect. **Increasing** the dose to 2% caused a slight retardation of the development of alopecia. However, a 60%

inhibition was observed at a topical dose of 5% minoxidil after 13 weeks of **treatment** ($p < 0.03$). The data demonstrate that **hair** loss in the AGA mouse is **androgen** dependent and that this mutant stain can serve as a suitable model for the screening of compounds, such as antiandrogens and vasodilators, which may influence the balding process.

ST HUMAN MINOXIDIL CYPROTERONE DERMATOLOGICAL-DRUG **TESTOSTERONE** DIHYDROTESTOSTERONE **THERAPEUTIC** DIET

RN 58-22-0 (TESTOSTERONE)
521-18-6 (DIHYDROTESTOSTERONE)
2098-66-0 (CYPROTERONE)
38304-91-5 (MINOXIDIL)

CC Biochemical Studies-General 10060
Biochemical Studies-Sterols and Steroids 10067
Pathology, General and Miscellaneous-Therapy *12512
Metabolism-Sterols and Steroids 13008
Nutrition-Prophylactic and Therapeutic Diets *13218
Endocrine System-General 17002
Endocrine System-Adrenals *17004
Endocrine System-Gonads and Placenta *17006
Integumentary System-Pathology *18506
Pharmacology-Clinical Pharmacology 22005
Pharmacology-Integumentary System, Dental and Oral Biology *22020
Laboratory Animals-General 28002

BC **Hominidae 86215**
Muridae 86375

L130 ANSWER 69 OF 97 HCAPLUS COPYRIGHT 1998 ACS

AN 1989:187398 HCAPLUS

DN 110:187398

TI Determination of cocaine, morphine, phenolbarbital, and methadone in cranial, acillary, and pubic **hair**

AU Balabanova, S.; Wolf, H. U.

CS Inst. Pathol. Rechtsmed., Univ. Ulm, Ulm, D-7900, Fed. Rep. Ger.

SO Laboratoriumsmedizin (1989), 13(2), 46-7
CODEN: LABOD3; ISSN: 0342-3026

DT Journal

LA German

CC 4-2 (Toxicology)
Section cross-reference(s): 1

AB The cocaine (I), methadone, morphine, and phenobarbital contents of the **hair** of habitual drug abusers, detd. by RIA, were the highest for pubic, followed by axillary, then cranial **hair**. The presence of I was also detectable in the axillary and pubic **hair** of a former drug user after 14 mo of abstinence. Results are discussed in relation to variations of **hair growth** rates with type.

ST drug abuse **hair** human addict; forensic drug abuse **hair** human

IT Legal chemistry and medicine
(drugs of abuse of **hair** of human addicts in)

IT **Hair**
(drugs of abuse of, of human addicts)

IT Pharmaceuticals
(of abuse, **hair** contents of, of human addicts)

IT **50-06-6**, Phenobarbital, biological studies 50-36-2, Cocaine 57-27-2, Morphine, biological studies 76-99-3, Methadone
RL: BIOL (Biological study)
(of axial and cranial and pubic **hair**, of drug addicts)

L130 ANSWER 70 OF 97 HCAPLUS COPYRIGHT 1998 ACS

AN 1990:146 HCAPLUS

DN 112:146

TI Sulfation of minoxidil in keratinocytes and **hair** follicles
AU Hamamoto, Tomoko; Mori, Yo
CS Dep. Biochem., Tokyo Coll. Pharm., Hachioji, 192-03, Japan
SO Res. Commun. Chem. Pathol. Pharmacol. (1989), 66(1), 33-44
CODEN: RCOCB8; ISSN: 0034-5164
DT Journal
LA English
CC 1-2 (Pharmacology)
AB Minoxidil, a potent antihypertensive agent, has the unique side effect of stimulating **hair growth**, and minoxidil sulfate may be the active form of minoxidil. Sulfation of minoxidil occurred in rat **hair** follicles and proliferative keratinocytes. In contrast, the activity in differentiating keratinocytes and fibroblasts was extremely low. The strong sulfation of minoxidil that occurred to **hair** follicle cells may be related to the **hair growth** -stimulating effect of this drug.
ST minoxidil sulfation **hair** follicle teratinocyte
IT **Hair**
(follicle, minoxidil sulfation by)
IT Skin, metabolism
(keratinocyte, minoxidil sulfation by)
IT 83701-22-8, Minoxidil sulfate
RL: FORM (Formation, nonpreparative)
(formation of, as minoxidil metabolite, in **hair** follicles and keratinocytes)
IT 9023-09-0, **Sulfotransferase**
RL: RCT (Reactant)
(of **hair** follicles and keratinocytes, minoxidil sulfation by)
IT 38304-91-5, Minoxidil
RL: RCT (Reactant)
(sulfation of, by **hair** follicles and keratinocytes)

L130 ANSWER 71 OF 97 HCAPLUS COPYRIGHT 1998 ACS
AN 1989:101527 HCAPLUS
DN 110:101527
TI Topical composition for **stimulating hair growth** with stable free radicals
IN Proctor, Peter H.
PA USA
SO PCT Int. Appl., 32 pp.
CODEN: PIXXD2
PI WO 8805653 A1 880811
DS W: AT, AU, BB, BG, BR, CH, DE, DK, FI, GB, HU, JP, KP, KR, LK, LU, MC, MG, MW, NL, NO, RO, SD, SE, SU
RW: AT, BE, BJ, CF, CG, CH, CM, DE, FR, GA, GB, IT, LU, ML, MR, NL, SE, SN, TD, TG
AI WO 88-US232 880127
PRAI US 87-8186 870128
DT Patent
LA English
IC ICM A61K007-06
ICS A61K031-625; A61K031-425; A61K031-495
CC 62-3 (Essential Oils and Cosmetics)
OS MARPAT 110:101527
AB The compn. contains, in an occlusive or semioclusive **pharmaceutical** carrier, a stable free radical-forming substance, such as minoxidil, a 5,5-diarylhydantoin, diazoxide, a porphyrin, proxyl, doxyl or tempo, an **antiandrogen** such as spironolactone, and optimally, a free radical scavenger such as DMSO, a tertiary phosphine oxide or a retinoid. The method involves applying the compn. to skin, preferably water-soaked skin, once or twice a day. A topical gel comprised 3 pt DMSO, 3 pt propylene

glycol, 3 pt H2O, 1% spironolactone, 1% diphenylhydantoin, and 1% hydroxypropyl cellulose.

ST **hair growth** stimulant free radical **antiandrogen**

IT Radicals, biological studies
RL: BIOL (Biological study)
(-forming substances, **hair growth** stimulants contg. **antiandrogens** and)

IT Retinoids
Sulfoxides
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**hair growth** stimulants contg.)

IT Nitroxides
RL: BIOL (Biological study)
(**hair growth** stimulants contg. **antiandrogens** and)

IT Carotenes and Carotenoids, biological studies
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**hair growth** stimulants contg., as free radical scavenger)

IT **Androgens**
RL: USES (Uses)
(**inhibitors, hair growth** stimulants contg.)

IT **Hair** preparations
(**growth** stimulants, contg. free radical-forming substances and **antiandrogens**)

IT 13840-40-9D, Phosphine oxide, tertiary derivs.
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**hair growth** stimulants contg.)

IT 57-41-0, 5,5-Diphenylhydantoin 364-98-7 586-96-9 723-57-9 917-95-3 1207-63-2 2154-68-9 2154-70-3 2226-96-2 2564-83-2D, derivs. 2896-70-0 3229-53-6D, derivs. 3229-73-0 3317-61-1 3376-24-7 4399-80-8 5389-27-5 6325-69-5 7772-37-4 10135-38-3 14559-54-7 14559-55-8 14691-88-4 15178-63-9 17932-40-0 21913-97-3 22690-04-6 24567-97-3 24799-67-5 24973-59-9 25554-61-4D, derivs. 25713-24-0 27048-01-7 29545-47-9 29545-48-0, 5-Doxylstearic acid 29639-21-2 31363-88-9 31363-89-0 36010-81-8 37157-85-0 37566-53-3 38568-24-0, Methyl 5-doxylstearate 39657-41-5 40293-62-7 40951-82-4, 7-Doxylstearic acid 50373-76-7 53034-38-1, 16-Doxylstearic acid 54060-41-2 54135-55-6 54606-49-4 56079-85-7 59719-53-8, Methyl 16-doxylstearate 61709-25-9 66641-27-8 66893-81-0 68407-07-8 68643-07-2 73283-40-6 73283-41-7 73283-43-9 73283-46-2 73283-48-4 73784-45-9 74648-17-2 76841-99-1 77695-02-4 78140-52-0 83016-63-1 84233-52-3 93003-12-4 95317-02-5 100900-11-6 100900-13-8 100900-39-8 100929-88-2 100929-91-7 100929-92-8 108321-38-6 119058-68-3 119058-69-4 119058-70-7 119164-01-1 119164-02-2 119164-03-3 119164-04-4
RL: BIOL (Biological study)
(**hair growth** stimulants contg. **antiandrogen** and)

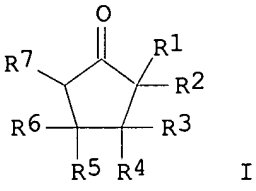
IT 359-85-3D, derivs. 461-72-3D, Hydantoin, diaryl derivs. 54976-00-0D, derivs. 119164-00-0D, derivs.
RL: BIOL (Biological study)
(**hair growth** stimulants contg. **antiandrogens** and)

IT 52-01-7, Spironolactone 427-51-0 2098-66-0
RL: BIOL (Biological study)
(**hair growth** stimulants contg. stable free radical-forming substance and)

IT 57-55-6, 1,2-Propanediol, biological studies 64-17-5, Ethanol, biological studies 67-56-1, Methanol, biological studies

67-68-5, biological studies 68-26-8, Retinol 71-23-8, Propanol, biological studies 71-36-3, Butanol, biological studies 79-80-1 79-81-2, Retinol palmitate 107-21-1, 1,2-Ethanediol, biological studies 116-31-4, Retinal 127-47-9, Retinyl acetate 302-79-4, Tretinoin 4759-48-2, Isotretinoin 5300-03-8, 9-cis-Tretinoin 29444-25-5 54350-48-0, Etretinate 73285-25-3 119164-05-5 119164-06-6
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**hair growth** stimulants contg., as free radical scavenger)

L130 ANSWER 72 OF 97 HCAPLUS COPYRIGHT 1998 ACS
AN 1989:236960 HCAPLUS
DN 110:236960
TI **Hair growth** stimulant containing cyclopentanone derivatives
IN Nakaguchi, Osamu; Kyotoo, Sumio; Ueno, Hiroshi; Takagi, Keiichi
PA Fujisawa Pharmaceutical Co., Ltd., Japan; V. Mane Fils Japan, Ltd.
SO Jpn. Kokai Tokkyo Koho, 4 pp.
CODEN: JKXXAF
PI JP 63275513 A2 881114 Showa
AI JP 87-111424 870506
DT Patent
LA Japanese
IC ICM A61K007-06
CC 62-3 (Essential Oils and Cosmetics)
Section cross-reference(s): 24, **63**
OS MARPAT 110:236960
GI



I

AB A **hair growth** stimulant contains cyclopentanone derivs. I (R1 = H, OH, alkyl, alkenyl, alkoxy; R2, R3 = H; R4 - R7 = H, lower alkyl; R1R2 may form alkylidene or alkenylidene; R2R3 may be a single bond). I **inhibit** the activity of **testosterone**-5.alpha.-reductase and **stimulate hair growth**. 2-(3,7-Dimethyl-6-octenylidene)cyclopentanone at 200 .mu.g/mL **inhibited** the activity of **testosterone**-5.alpha.-reductase in a homogenate of rat prostate gland by 72.0%. A hair prepn. contg. 1-methylcyclopenten-3-one 0.5, capronium chloride 1.0, 95% EtOH 48.0, H2O 50.0, vitamin E 0.5%, a flavor, a coloring material, and an antiseptic was prepd.
ST cyclopentanone deriv **hair growth** stimulant; **testosterone** reductase **inhibitor hair growth**
IT Hair preparations
(tonics, contg. cyclopentenone derivs. as **testosterone** -reductase **inhibitors**)
IT 80-71-7 95-41-0 930-30-3, 2-Cyclopenten-1-one 931-22-6 1120-73-6 1128-08-1 2758-18-1 16424-41-2 25564-22-1 28790-86-5 30434-64-1 30434-65-2 30434-70-9 54458-61-6 64351-95-7 68043-00-5 68922-13-4 77342-87-1 120393-42-2 120393-43-3 120995-61-1

RL: BIOL (Biological study)
(hair tonics contg.)
IT 9036-43-5, **Testosterone**-5.alpha.-reductase
RL: BIOL (Biological study)
(**inhibitors** for, cyclopentenone derivs. as, hair tonics contg.)

L130 ANSWER 73 OF 97 BIOSIS COPYRIGHT 1998 BIOSIS
AN 88:507985 BIOSIS
DN BA86:128669
TI DIFFERENTIAL SUPPRESSION OF **TESTOSTERONE** AND ESTRADIOL IN HIRSUTE WOMEN WITH THE SUPERACTIVE GONADOTROPIN-RELEASING HORMONE AGONIST LEUPROLIDE.
AU RITTMASTER R S
CS DEP. MED., HALIFAX INFIRMARY, 1335 QUEEN STREET, HALIFAX, NOVA SCOTIA, CANADA B3J 2H6.
SO J CLIN ENDOCRINOL METAB 67 (4). 1988. 651-655. CODEN: JCEMAZ ISSN: 0021-972X
LA English
AB To determine the dose of the GnRH agonist leuprolide necessary to maximally suppress ovarian **testosterone** secretion, 10 moderately to severely hirsute women (5 with idiopathic hirsutism and 5 with polycystic ovarian syndrome) were given gradually **increasing** leuprolide doses, starting with either 5 or 10 .mu.g/kg .cntdot. day. Serum **testosterone** and estradiol, basal LH, and the LH response to GnRH were measured before and at the end of each **treatment** period, until maximal suppression of estradiol and **testosterone** occurred. Leuprolide was then continued for a total of 6 months to assess its clinical efficacy. Hirsutism scores and **hair growth** rates were determined before and after **therapy**. Serum estradiol and the LH response to GnRH were maximally or near-maximally suppressed in all women by the lowest doses of leuprolide used. Basal serum LH was not maximally suppressed in all women until a dose of 15 .mu.g/kg .cntdot. day was reached, and maximal **testosterone** suppression required 15 .mu.g/kg .cntdot. day or more in 7 of the 10 women. The addition of 0.5 mg dexamethasone daily for 4 weeks at the end of the study in 5 of the women **reduced** serum **testosterone** to undetectable levels. Symptomatic improvement in hirsutism occurred in 9 women, hirsutism scores decreased by at least 3 points in 5 women, and **hair growth** rates decreased in 8 women. These data indicate that low doses of leuprolide were sufficient to maximally suppress serum estradiol and the LH response to exogenous GnRH. Higher leuprolide doses were needed to maximally suppress serum **testosterone** and the basal LH level. Leuprolide (20 .mu.g/kg .cntdot. day) effectively **reduced hair growth** in the majority of these women.
ST HORMONE-DRUG DERMATOLOGICAL-DRUG LUTEINIZING HORMONE POLYCYSTIC OVARY SYNDROME
RN 50-28-2 (ESTRADIOL)
58-22-0 (TESTOSTERONE)
9002-67-9 (LUTEINIZING HORMONE)
53714-56-0 (LEUPROLIDE)
CC Clinical Biochemistry; General Methods and Applications *10006
Biochemical Studies-Proteins, Peptides and Amino Acids 10064
Biochemical Studies-Sterols and Steroids 10067
Biochemical Studies-Carbohydrates 10068
Metabolism-Carbohydrates 13004
Metabolism-Sterols and Steroids *13008
Metabolism-Proteins, Peptides and Amino Acids *13012
Food Technology-Cereal Chemistry 13510
Reproductive System-Pathology *16506
Endocrine System-Gonads and Placenta *17006

Endocrine System-Pituitary *17014
Endocrine System-Neuroendocrinology *17020
Integumentary System-Pathology *18506
Nervous System-Pathology 20506
Pharmacology-Clinical Pharmacology *22005
Pharmacology-Endocrine System *22016
Pharmacology-Integumentary System, Dental and Oral Biology *22020
Pharmacology-Reproductive System; Implantation Studies *22028
Developmental Biology-Embryology-Descriptive Teratology and Teratogenesis 25552
BC **Hominidae 86215**

L130 ANSWER 74 OF 97 WPIDS COPYRIGHT 1998 DERWENT INFORMATION LTD
AN 87-321912 [46] WPIDS
DNC C87-137213
TI Synergistic compsn. contg. minoxidil and cyproterone acetate - with synergistic activity for **reducing hair** loss and improving **hair growth**.
DC B01 B03
IN LIMAT, A; NOSER, F
PA (WELA) WELLA AG
CYC 1
PI DE 3615396 A 871112 (8746)* 4 pp
ADT DE 3615396 A DE 86-3615396 860507
PRAI DE 86-3615396 860507
IC **A61K007-06**
AB DE 3615396 A UPAB: 930922
Compsn. for treating the hair and scalp contains, apart from usual cosmetic carriers and additives, a mixt of minoxidil (I; 2,6-diamino-4-piperidino pyrimidine-1-oxide) and cyproterone acetate (II;17-acetoxy 6-chloro-1alpha,2alpha methylene-4,6-pregnodiene 3,20-dione).
Compsns. pref. contain 0.01-5 wt.% (I) and 0.01-2 wt.% (II) esp. totalling 0.2-5 wt.%.
USE/ADVANTAGE - The compsn. **reduce hair** loss and **stimulate hair growth**. (II) is a known **antiandrogenic** agent and (II) is already known to improve **hair growth** in some subjects. When used together, these cpds. have a synergistic effect, i.e. they visibly improve **hair growth** in at least 70% of those treated.
0/0
FS CPI
FA AB; DCN
MC CPI: B01-C03; B07-D05; B07-D12; B12-C09; B12-G01A; B12-L05

L130 ANSWER 75 OF 97 BIOSIS COPYRIGHT 1998 BIOSIS
AN 88:158768 BIOSIS
DN BA85:82421
TI **HAIR GROWTH** AND **ANDROGEN** RESPONSES IN HIRSUTE WOMEN **TREATED** WITH CONTINUOUS CYPROTERONE ACETATE AND CYCLICAL ETHYNYLESTRADIOL.
AU JONES D B; IBRAHAM I; EDWARDS C R W
CS DEP. MED., WESTERN GEN. HOSP., CREWE ROAD SOUTH, EDINBURGH EH4 2XU, SCOTLAND.
SO ACTA ENDOCRINOL 116 (4). 1987. 497-501. CODEN: ACENA7 ISSN: 0001-5598
LA English
AB Eighteen hirsute women (8 with polycystic ovarian syndrome, 10 with idiopathic hirsutism) were **treated** for up to 12 months with cyproterone acetate, 150 mg daily, and ethinyl estradiol, 50 .mu.g on days 5-25 of the menstrual cycle. **Hair growth**

rate and density were measured from standardized serial photographs of a shaved skin area. A significant **reduction** was seen in mean **hair growth** rate, total plasma **testosterone**, free **testosterone** index, plasma dehydroepiandrosterone, and plasma androstenedione. LH and FSH also fell and sex hormone binding globulin level **increased**. No significant changes occurred in **hair** density or in serum PRL levels. A significant correlation was observed between **hair growth** rate and total plasma **testosterone** for the pooled results ($r = 0.35$, $P < 0.005$). No significant correlations were seen between **hair** density and the endocrine parameters studied.

ST POLYCYSTIC OVARIAN SYNDROME DERMATOLOGICAL-DRUG HORMONE-DRUG **TESTOSTERONE** DEHYDROEPIANDROSTERONE LUTEINIZING HORMONE FSH ANDROSTENEDIONE SEX HORMONE BINDING GLOBULIN LEVEL PHARMACODYNAMICS DRUG-DRUG INTERACTION PHOTOGRAPHY

RN 53-43-0 (DEHYDROEPIANDROSTERONE)
57-63-6 (ETHYNYLESTRADIOL)
58-22-0 (TESTOSTERONE)
63-05-8 (ANDROSTENEDIONE)
427-51-0 (CYPROTERONE ACETATE)
9002-67-9 (LUTEINIZING HORMONE)
9002-68-0 (FSH)

CC Methods, Materials and Apparatus, General-Photography 01012
Biochemical Studies-Proteins, Peptides and Amino Acids 10064
Biochemical Studies-Sterols and Steroids 10067
Pathology, General and Miscellaneous-Therapy 12512
Metabolism-Sterols and Steroids *13008
Metabolism-Proteins, Peptides and Amino Acids *13012
Reproductive System-Pathology *16506
Endocrine System-Gonads and Placenta *17006
Endocrine System-Pituitary *17014
Integumentary System-Pathology *18506
Pharmacology-Drug Metabolism; Metabolic Stimulators *22003
Pharmacology-Clinical Pharmacology *22005
Pharmacology-Endocrine System *22016
Pharmacology-Integumentary System, Dental and Oral Biology *22020
Pharmacology-Reproductive System; Implantation Studies *22028

BC **Hominidae 86215**

L130 ANSWER 76 OF 97 BIOSIS COPYRIGHT 1998 BIOSIS

AN 87:195296 BIOSIS

DN BA83:103420

TI LONG-TERM TREATMENT WITH SODIUM VALPROATE MONITORING OF VENOUS AMMONIA CONCENTRATIONS AND ADVERSE EFFECTS.

AU ZACCARA G; CAMPOSTRINI R; PAGANINI M; MESSORI A; VALENZA T; ARNETOLI G; ZAPPOLI R

CS 2ND NEUROLOGICAL INST., UNIV. FLORENCE, VIALE MORGAGNI 85, 50134 FLORENCE, ITALY.

SO THER DRUG MONIT 9 (1). 1987. 34-40. CODEN: TDMODV ISSN: 0163-4356

LA English

AB Adverse effects and venous blood ammonia concentrations were monitored over a period of 7 months in patients with epilepsy treated with valproate (VPA). During the 1st, 4th, 12th, 20th, nd 28th weeks of therapy, blood samples for analysis of ammonia and anticonvulsants were taken immediately before the morning dose of VPA as well as 2 h after dosing. In all, 40 patients completed the follow-up; 16 of these (Group 1) received VPA alone, while the remaining 24 (Group 2) were treated simultaneously with VPA and other anticonvulsants (phenobarbital, phenytoin, and/or carbamazepine). In Group 1 patients, a slight though significant increase in ammonia concentrations was found during long-term VPA treatment; this trend

was even more pronounced in Group 2 patients. The difference between postdose and predose ammonia levels in Group 2 patients was significant at each of the five follow-up examinations. In contrast, no such difference was demonstrated in patients of Group 1. VPA concentrations were found to be consistently higher in Group 2 patients than in Group 1. Twenty-three patients complained of various long-term adverse effects, while the other 17 remained symptom-free. The adverse effects reported included drowsiness, tremors, weight gain, **hair** loss, and gastrointestinal symptoms. Our data confirm the previously suggested hypothesis that changes in venous blood ammonia are particularly evident in patients taking VPA in combination with other antiepileptic drugs, such as phenobarbital and phenytoin.

ST HUMAN PHENOBARBITAL PHENYTOIN CARBAMAZEPINE EPILEPSY PHARMACOKINETICS DROWSINESS TREMORS WEIGHT GAIN **HAIR** LOSS GASTROINTESTINAL SYMPTOMS

RN **50-06-6** (PHENOBARBITAL)
57-41-0 (PHENYTOIN)
298-46-4 (CARBAMAZEPINE)
1069-66-5 (SODIUM VALPROATE)
7664-41-7 (AMMONIA)

CC Biochemical Studies-General 10060
Biochemical Studies-Proteins, Peptides and Amino Acids 10064
Pathology, General and Miscellaneous-Diagnostic 12504
Pathology, General and Miscellaneous-Therapy 12512
Nutrition-Malnutrition; Obesity 13203
Digestive System-Pathology 14006
Muscle-Pathology 17506
Integumentary System-Pathology 18506
Nervous System-Pathology *20506
Psychiatry-Psychophysiology 21003
Pharmacology-Drug Metabolism; Metabolic Stimulators *22003
Pharmacology-Clinical Pharmacology *22005
Pharmacology-Neuropharmacology *22024
Toxicology-Pharmacological Toxicology *22504

BC **Hominidae 86215**

L130 ANSWER 77 OF 97 MEDLINE

AN 87217926 MEDLINE

DN 87217926

TI Zinc status and delayed cutaneous hypersensitivity in handicapped children treated with anticonvulsants.

AU Higashi A; Chen C; Matsuda I

SO DEVELOPMENTAL PHARMACOLOGY AND THERAPEUTICS, (1987) 10 (1) 30-5. Journal code: EAF. ISSN: 0379-8305.

CY Switzerland

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 198709

AB Delayed cutaneous hypersensitivity and hair zinc contents were investigated in 68 children treated with anticonvulsants and 14 untreated children, and serum zinc contents were also measured in 21 of the treated and 13 of the untreated children. Serum zinc levels in the treated and untreated children were 82.7 +/- 7.1 and 85.1 +/- 18.2 micrograms/dl, respectively. Hair zinc levels in the treated and untreated children were 145.4 +/- 27.0 and 144.3 +/- 20.1 micrograms/g, respectively. These two parameters were not significantly different between the two groups. However, a significantly depressed skin reaction and a higher incidence of hypozincnemia (below 70 micrograms/dl) were found in the treated children (p less than 0.05). The results indicated that phenytoin-induced zinc deficiency might be one of the possible factors or exacerbatory factors in suppressed cellular immunity

found with anticonvulsant therapy.
CT Check Tags: Female; Human; Male
Adolescence
Child
Child, Preschool
Dinitrochlorobenzene: IM, immunology
Disabled Persons
*Drug Hypersensitivity: ET, etiology
Hair: AN, analysis
*Hypersensitivity, Delayed: CI, chemically induced
*Phenobarbital: AE, adverse effects
Phenobarbital: TU, therapeutic use
*Phenytoin: AE, adverse effects
Phenytoin: TU, therapeutic use
Skin Tests
*Zinc: AN, analysis
Zinc: DF, deficiency
RN **50-06-6 (Phenobarbital)**; 57-41-0 (Phenytoin); 7440-66-6 (Zinc); 97-00-7 (Dinitrochlorobenzene)

L130 ANSWER 78 OF 97 WPIDS COPYRIGHT 1998 DERWENT INFORMATION LTD
AN 86-252147 [38] WPIDS
DNC C86-108674
TI Use of melatonin compsns. - for treating acne vulgaris, seborrhoea, hirsutism and for rejuvenation of hair follicles.
DC D21 E13
IN PIERPAOLI, W; REGELSON, W
PA (CELL-N) CELLENA CELL ENG AG; (CELL-N) CELLENA CELL ENGENEERING AG
CYC 16
PI WO 8605093 A 860912 (8638)* EN 41 pp
RW: AT BE CH DE FR GB IT LU NL SE
W: AU DK JP
AU 8656267 A 860924 (8650)
EP 214254 A 870318 (8711) EN
R: AT BE CH DE FR GB IT LI LU NL SE
JP 62502118 W 870820 (8739)
DK 8605221 A 861031 (8749)
US 4746674 A 880524 (8823)
CA 1292947 C 911210 (9205)
EP 214254 B1 920617 (9225) EN 14 pp A61K007-06 <--
R: AT BE CH DE FR GB IT LI LU NL SE
DE 3685696 G 920723 (9231) A61K007-06 <--
JP 07078007 B2 950823 (9538) 12 pp A61K007-00 <--
DK 170513 B 951009 (9546) A61K007-06 <--
ADT WO 8605093 A WO 86-EP108 860303; EP 214254 A EP 86-901842 860303; US 4746674 A US 85-770054 850827; EP 214254 B1 EP 86-901842 860303, WO 86-EP108 860303; DE 3685696 G DE 86-3685696 860303, EP 86-901842 860303, WO 86-EP108 860303; JP 07078007 B2 JP 86-501583 860303, WO 86-EP108 860303; DK 170513 B WO 86-EP108 860303, DK 86-5221 861031
FDT EP 214254 B1 Based on WO 8605093; DE 3685696 G Based on EP 214254, Based on WO 8605093; JP 07078007 B2 Based on JP 62502118, Based on WO 8605093; DK 170513 B Previous Publ. DK 8605221
PRAI GB 85-5537 850304; US 85-770054 850827
REP No-Citns. ; 2.Jnl.Ref ; EP 126630
IC ICM **A61K007-06**
ICS **A61K007-48; A61K031-40; A61K031-405** ; C07D209-16
AB WO 8605093 A UPAB: 930922
Improvement in the cosmetic and physical appearance of skin is effected by topical admin. of a compsn. of melatonin (I) and a carrier. (I) enhances the local action of oestrogen and attenuates the systemic action of anhydrogens at the site administered.
The method is claimed for (1) treating acne vulgaris or seborrhoea; (2) selectively **decreasing** body and facial

hair growth by attenuating the **stimulation** of oestrogen induced **hair growth**; and (3) **reducing** excessive **hair** fall where the air follicles are not degenerated and can be made to grow.
The compsn. is pref. applied in the evening prior to sleeping when endogenous (I) prodn. is at a low level.
0/0
FS CPI
FA AB
MC CPI: D08-B09A; E06-D01

L130 ANSWER 79 OF 97 WPIDS
AN 86-119079 [18] WPIDS
DNC C86-050768
TI **Hair growth** modification - by topical application of a material **inhibiting** the action of ornithine decarboxylase.
DC B01 B05 D21
IN SHANDER, D
PA (HAND-I) HANDELMAN J H; (SHAN-I) SHANDER D
CYC 21
PI WO 8602269 A 860424 (8618)* EN 18 pp
RW: AT BE CH DE FR GB IT LU NL SE
W: AU DK JP NO
ZA 8507846 A 860414 (8628)
AU 8548673 A 860502 (8630)
EP 198893 A 861029 (8644) EN
R: AT BE CH DE FR GB IT LI LU NL SE
NO 8602339 A 860915 (8644)
CN 85108498 A 860610 (8710)
JP 62500932 W 870416 (8721)
DK 8602784 A 860613 (8722)
US 4720489 A 880119 (8805)
CA 1262335 A 891017 (8947)
EP 198893 B 920304 (9210)
R: AT BE CH DE FR GB IT LI LU NL SE
DE 3585526 G 920409 (9216)
NZ 213805 A 930428 (9320) A61K007-06 <--
DK 166801 B 930719 (9334) A61K007-06 <--
NO 174832 B 940411 (9418) A61K031-56 <--
JP 06053680 B2 940720 (9427) 5 pp A61K045-00 <--
PH 26283 A 920410 (9520) A61K031-165 <--
ADT WO 8602269 A WO 85-US2000 851010; ZA 8507846 A ZA 85-7846 851011; EP 198893 A EP 85-905536 851010; JP 62500932 W JP 85-504753 851010; US 4720489 A US 84-661019 841015; NZ 213805 A NZ 85-213805 851014; DK 166801 B WO 85-US2000 851010, DK 86-2784 860613; NO 174832 B WO 85-US2000 851010, NO 86-2339 860611; JP 06053680 B2 JP 85-504753 851010, WO 85-US2000 851010; PH 26283 A PH 85-32920 851011
FDT DK 166801 B Previous Publ. DK 8602784; NO 174832 B Previous Publ. NO 8602339; JP 06053680 B2 Based on JP 62500932, Based on WO 8602269
PRAI US 84-661019 841015
REP DE 2840144; EP 16239; SSR880629 ; US 4201788; US 4390532; US 4439432; US 4457925; 3.Jnl.Ref ; US 4456586
IC **A61K007-06; A61K031-56; A61K045-00**
ICM **A61K007-06; A61K031-165; A61K031-56 ; A61K045-00**
ICS **A61K031-13; A61K031-195; A61K031-565; A61K031-57; A61K037-48**
ICI **A61K031-13, A61K031:**
AB WO 8602269 A UPAB: 930922
A process of altering the rate and character of human **hair growth** comprises applying to the skin a compsn. contg. a material capable of **inhibiting** the action of the enzyme ornithine decarboxylase (ODC). The compsn. may contain e.g.

2-(difluoromethyl)-2,5 -diaminopentanoic acid; alpha-ethynyl ornithine, 6-heptyne-2,5-diamine or 2-methyl-6-heptyne diamine. Prefd. application rate of the material is 50-500 microgram/sq.cm.
The compsn. may also contain an anti-**androgen** material selected from 5-alpha-**reductase** **inhibitors** and cytoplasmic **androgen** receptor-binding agents.
USE/ADVANTAGE - The rate and character of human **hair** **growth**, including male beard **hair growth** , can be altered. Unwanted interference with other bodily processes can be minimised or avoided.
0/0

FS CPI
FA AB
MC CPI: B01-C04; B01-C05; B04-B04F; B04-C03D; B10-B01B; B10-B02J; B10-E04C; B12-G01A; D08-B

L130 ANSWER 80 OF 97 BIOSIS COPYRIGHT 1998 BIOSIS
AN 86:458098 BIOSIS
DN BA82:114940
TI **TREATMENT** OF HIRSUTISM WITH A GONADOTROPIN-RELEASING HORMONE AGONIST NAFARELIN.
AU ANDREYKO J L; MONROE S E; JAFFE R B
CS REPRODUCTIVE ENDOCRINOL. CENT., DEP. OBSTETRICS GYNECOL., REPRODUCTIVE SCI., UNIV. CALIFORNIA, SAN FRANCISCO, CALIF. 94143.
SO J CLIN ENDOCRINOL METAB 63 (4). 1986. 854-859. CODEN: JCEMAZ ISSN: 0021-972X
LA English
AB GnRH analoges **inhibit** the secretion of gonadotropins and, therefore, that of estrogens and **androgens** of ovarian origin. The purpose of this study was to investigate the use of one superactive agonistic GnRH analog, nafarelin, in the **treatment** of hirsutism. Six hirsute women were **treated** with nafarelin (1000 .mu.g/day) for 6 months. An acute rise in serum gonadotropin levels occurred in response to nafarelin administration initially, but it lasted less than 2 weeks. Serum gonadotropin, **testosterone**, free **testosterone**, and androstenedione concentrations decreased significantly during **treatment**. Mean serum LH levels decreased from 17.9 .+-. 4.6 (.+-.SE) to 5.0 .+-. 0.5 mIU/ml (P < 0.01), and FSH decreased from 9.3 .+-. 0.7 to 7.2 .+-. 0.9 mIU/ml (P < 0.05) after 1 month of **treatment**. The total **testosterone** concentration fell from 0.77 .+-. 0.10 to 0.40 .+-. 0.14 ng/ml (P < 0.01) after 1 month of **therapy**, and free **testosterone** decreased from 10.7 .+-. 2.7 to 4.1 .+-. 1.6 pg/ml (P < 0.01) after 3 months. Androstenedione levels decreased from 2.4 .+-. 0.4 to 1.2 .+-. 0.2 ng/ml (P < 0.01) after 1 month of **treatment**. The mean concentrations of all of the above hormones remained suppressed throughout **treatment**. Serum 5.alpha.-androstane-3.alpha.,17.beta.-diol glucuronide levels did not decrease significantly during **treatment**, nor did dehydroepiandrosterone sulfate levels. The mean estradiol concentration during **treatment** was 34.8 .+-. 3.1 pg/ml. The clinical response was very good; **hair growth** was slower, and new **hair** was less coarse compared to the pretreatment period. Hirsutism scores (determined by Ferriman-Gallwey assessment of extent and quality of body **hair**) improved in four of the six patients. In the six patients, the mean score decreased significantly from 19.3 .+-. 3.3 to 13.2 .+-. 2.8 (P < 0.05) at the end of **treatment**. These data demonstrate that by suppressing ovarian **androgen** production, nafarelin may be useful for the **treatment** of hirsutism associated with either **increased** ovarian **androgen** production or **increased** sensitivity of the **hair** follicle to

normal concentrations of circulating **androgens.**
ST HUMAN METABOLIC-DRUG **TESTOSTERONE** ANDROSTENEDIONE LUTEINIZING HORMONE 5-ALPHA ANDROSTANE-3-ALPHA 17-BETA-DIOL GLUCURONIDE DEHYDROEPIANDROSTERONE
RN 53-43-0 (DEHYDROEPIANDROSTERONE)
58-22-0 (TESTOSTERONE)
63-05-8 (ANDROSTENEDIONE)
9002-67-9 (LUTEINIZING HORMONE)
76932-56-4 (NAFARELIN)
CC Biochemical Studies-Proteins, Peptides and Amino Acids 10064
Biochemical Studies-Sterols and Steroids 10067
Biochemical Studies-Carbohydrates 10068
Pathology, General and Miscellaneous-Therapy *12512
Metabolism-Carbohydrates 13004
Metabolism-Sterols and Steroids *13008
Metabolism-Proteins, Peptides and Amino Acids *13012
Blood, Blood-Forming Organs and Body Fluids-Blood and Lymph Studies 15002
Endocrine System-Gonads and Placenta *17006
Endocrine System-Pituitary *17014
Endocrine System-Neuroendocrinology *17020
Integumentary System-Pathology *18506
Nervous System-Physiology and Biochemistry *20504
Pharmacology-Drug Metabolism; Metabolic Stimulators *22003
Pharmacology-Clinical Pharmacology *22005
Pharmacology-Endocrine System *22016
Pharmacology-Integumentary System, Dental and Oral Biology *22020
BC **Hominidae 86215**

L130 ANSWER 81 OF 97 WPIDS COPYRIGHT 1998 DERWENT INFORMATION LTD
AN 85-159178 [26] WPIDS
CR 90-044777 [06]
DNC C85-069670
TI Topical compsn. contg. anti-**androgen**(s) - for altering rate and character of **androgen-stimulated hair growth.**
DC B01 B05
IN BREUER, M M; SHANDER, D; USDIN, R V; VAN, DER LEE H; KASZYNSKI, E; USDIN, V R; KASZYNSKI, E G
PA (KASZ-I) KASZYNSKI E G; (HAND-I) HANDELMAN J H; (KASZ-I) KASZYNSKY E G; (KASZ-I) KASZUNSKI E G
CYC 16
PI WO 8502543 A 850620 (8526)* EN 15 pp
RW: CH DE FR GB NL SE
W: AU DK JP NO
AU 8537458 A 850626 (8536)
ZA 8409518 A 850612 (8536)
NO 8503143 A 851014 (8548)
EP 165970 A 860102 (8602) EN
R: CH DE FR GB LI NL SE
JP 61500966 W 860515 (8626)
DK 8503630 A 850809 (8632)
CN 85101410 A 870110 (8806)
CA 1251737 A 890328 (8917)
CN 1047620 A 901212 (9136)#
IT 1221006 B 900621 (9216)
EP 165970 B1 930303 (9309) EN 9 pp A61K031-56 <--
R: CH DE FR GB LI NL SE
DE 3486090 G 930408 (9315) A61K031-56 <--
PH 26282 A 920410 (9520) A61K031-56 <--
JP 07045382 B2 950517 (9524) 6 pp A61K007-06 <--
DK 170726 B 951227 (9606) A61K031-56 <--
ADT WO 8502543 A WO 84-US1977 841130; ZA 8409518 A ZA 84-9518 841206; EP

165970 A EP 85-900364 841130; JP 61500966 W JP 85-500023 841130; EP 165970 B1 WO 84-US1977 841130, EP 85-900364 841130; DE 3486090 G DE 84-3486090 841130, WO 84-US1977 841130, EP 85-900364 841130; PH 26282 A PH 84-31556 841210; JP 07045382 B2 WO 84-US1977 841130, JP 85-500023 841130; DK 170726 B WO 84-US1977 841130, DK 85-3630 850809
FDT EP 165970 B1 Based on WO 8502543; DE 3486090 G Based on EP 165970, Based on WO 8502543; JP 07045382 B2 Based on JP 61500966, Based on WO 8502543; DK 170726 B Previous Publ. DK 8503630
PRAI US 83-560726 831212; US 85-807623 851211
REP DE 2840144; SSR871104 ; US 4008802; US 4039669; US 4269831; US 4310523; US 4439432; US 4098802
IC ICM **A61K007-06; A61K031-56**
ICS **A61K007-15; A61K031-555; A61K037-43**
AB WO 8502543 A UPAB: 950619
A topical compsn. for altering the rate and character of **androgen-stimulated hair growth** comprises at least one 5-alpha-**reductase inhibitor** (I) and/or cytoplasmic **androgen** receptor-binding agent (II), and a suitable carrier.
USE - The normal rate of mole beard **hair growth** is **reduced** and its character caused to pevert toward the vellus state by the topical application of (I) and/or (II). By the proper selection of anti-**androgen** cpds. and their mode of use, unwanted interference with other **androgen**-mediated bodily processes can be minimised or avoided.
0
Dwg./0
FS CPI
FA AB
MC CPI: B01-C04; B01-C05; B01-C09; B01-D01; B06-D18; B10-F02; B12-A07; B12-G01; B12-L05

L130 ANSWER 82 OF 97 WPIDS COPYRIGHT 1998 DERWENT INFORMATION LTD
AN 85-261677 [42] WPIDS
DNN N85-195547 DNC C85-113530
TI Treatment of hirsutism with hormonal preparate - involves one monthly per-cutaneous injection of **testosterone** -propionate.
DC B01
IN ABOVYAN, M S; DOLYAN, G G; KHACHIKYAN, M A
PA (OBST-R) OBSTETRICS GYNECOLO
CYC 1
PI SU 1148620 A 850407 (8542)* 2 pp
ADT SU 1148620 A SU 79-2729634 790228
PRAI SU 79-2729634 790228
IC **A61K037-24**
AB SU 1148620 A UPAB: 930925
The injections are given regardless of the menstrual cycle using 5% **testosterone** propionate soln. The dose is **increased** from 0.02 to 0.06 ml over a period of 3-4 months. As previously, the treatment involves administration of hormonal preparates.
USE/ADVANTAGE - **Increased** therapeutic effect and prevention of side effects in medical practice, esp. gynaecological endocrinology.
In an example, a 16 year old patient with Schtein-Levental syndrome was treated by the proposed method. After 1 month pathological **growth** of **hair** disappeared from the face, nipples, stomach and the small of the back. Hypertrichosis of the extremities was considerably **reduced**. No side effects were noticed. Bul.13/7.4.85
0/0
FS CPI
FA AB

MC CPI: B01-C05; B12-G04; B12-M07

L130 ANSWER 83 OF 97 WPIDS COPYRIGHT 1998 DERWENT INFORMATION LTD
AN 85-113073 [19] WPIDS
DNC C85-048879
TI Cosmetic materials for **stimulating hair growth** and treating acne - contain 4-oestren(3)one-17-beta-ethoxy deriv(s).
DC B01 D21 E19
PA (SHIS) SHISEIDO CO LTD
CYC 1
PI JP 60054310 A 850328 (8519)* 9 pp
ADT JP 60054310 A JP 83-162944 830905
PRAI JP 83-162944 830905
IC **A61K007-06**; **A61K031-56**; C07J001-00; C07J017-00; C07J031-00; C07J043-00
AB JP60054310 A UPAB: 930925
Cosmetic materials contain at least one species of 4-oestren-3-one-17 beta-ethoxy derivs. of formula (I) (R is -CH2OH, -CH2OCOX (X is 1-5C alkyl or phenyl), -CH2Y (Y is F, Cl, Br or I), -CH2CN, -COOX -CH2OSO2C6H4CH3, -CH2OSO2CH3, (a) or (b).
ADVANTAGE - Cosmetics have no undesirable side-effects such as hormone action, and both **inhibit reductase** activity and **inhibit** combination of 5 alpha **dihydrotestosterone** and receptor protein. They are excellent in **hair-growing** effect and curing acne.
0/0
FS CPI
FA AB
MC CPI: B01-C05; B12-A07; B12-G01; B12-L02; D08-B03; E01

L130 ANSWER 84 OF 97 HCAPLUS COPYRIGHT 1998 ACS DUPLICATE 8
AN 1985:466296 HCAPLUS
DN 103:66296
TI Hepato- and neurotoxicity by ethylenthiourea
AU Ugazio, G.; Brossa, O.; Grignolo, F.
CS Fac. Med. Surg., Univ. Torino, Turin, I-10125, Italy
SO Res. Commun. Chem. Pathol. Pharmacol. (1985), 48(3), 401-14
CODEN: RCOCB8; ISSN: 0034-5164
DT Journal
LA English
CC 4-3 (Toxicology)
AB The toxicity of ETU [96-45-7] to nonthyroid tissues and the possible enhancement of the toxicity by drugs or other chems. were studied in rats. In chronic administration, ETU toxicity was higher in male than in female rats. Simultaneous administration of ETU with EtOH [64-17-5] increased ETU toxicity, whereas phenobarbital [**50-06-6**] decreased ETU toxicity. Liver secretion of triglycerides was impaired by ETU acute administration, which resulted in steatosis. This was not obsd. during subacute administration. Liver microsomal cytochrome P 450 [9035-51-2] was reduced after a long-term administration. After 30-wk treatment, 28.6% of the animals died; prolonged **growth** retardation, **alopecia** (80% loss of **hair**), severe conjunctivitis, blepharitis, and peripheral nervous system disorders were obsd. in survivors. Thus, relations should be studied between hazardous compds. and possible potentiating factors; organs not yet recognized as targets should be also studied when setting tolerance limits for ambient pollution.
ST ETU hepatotoxicity neurotoxicity sex ethanol; alc phenobarbital ETU hepatotoxicity
IT Sex
(ETU toxicity in relation to)

IT Liver, toxic chemical and physical damage
(ETU toxicity to, sex in relation to)
IT **Alopecia**
(from ETU)
IT Eye, toxic chemical and physical damage
(blepharitis, from ETU, sex in relation to)
IT Eye, toxic chemical and physical damage
(conjunctivitis, from ETU, sex in relation to)
IT Nervous system
(peripheral, disease, injury, from ETU, sex in relation to)
IT **50-06-6**, biological studies 64-17-5, biological studies
RL: BIOL (Biological study)
(ETU toxicity response to, sex in relation to)
IT 9035-51-2, biological studies
RL: BIOL (Biological study)
(of liver microsome, ETU effect on)
IT 96-45-7
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(toxicity of, to liver and peripheral nervous system, ethanol and phenobarbital effect on, sex in relation to)

L130 ANSWER 85 OF 97 WPIDS COPYRIGHT 1998 DERWENT INFORMATION LTD
AN 84-279239 [45] WPIDS
DNC C84-118568
TI **Hair growth** stimulant - comprising capronium chloride, at least one female hormone, e.g. ethynyl oestradiol, and opt. **testosterone** 5-alpha **reductase inhibitor.**
DC B01 B05 D21 E19
PA (SHIS) SHISEIDO CO LTD
CYC 1
PI JP 59172412 A 840929 (8445)* 5 pp
ADT JP 59172412 A JP 83-46934 830319
PRAI JP 83-46934 830319
IC **A61K007-06**
AB JP59172412 A UPAB: 930925
Hair-growth stimulant (I) contains capronium chloride (II) and at least one female hormone (III) and opt. at least one **testosterone**-5-alpha-**reductase inhibitor** (IV).
(III) may be ethynyl oestradiol, 17beta-oestradiol, oestriol and oestrone. (IV) is e.g. androstenedione, 4-androsten-3-one 17beta-carboxylic acid, progesterone, corticosterone or hydrocortisone.
(II) is methyl-N-trimethyl- gamma-aminobutyrate chloride. (II), (III) and (IV) are used pref. in 0.1-5 (esp. 0.1-2), 0.0001-0.005 and 0.001-2 wt% to (I). (I) may also contain e.g. an agent such as vitamin E, benzyl nicotinate, vitamin A, biotin and menthol, oil such as olive oil, squalane and higher alcohol, surfactant, antioxidant and water.
ADVANTAGE - Material can exert a much elevated **hair-growing** effect without producing unwanted side effects, esp. due to female hormones.
0/0
FS CPI
FA AB
MC CPI: B01-A01; B01-A02; B01-C02; B01-C04; B01-C05; B01-C09; B10-A22; B12-G01; B12-G04; B12-L05; D08-B03; E01; E10-A22

L130 ANSWER 86 OF 97 WPIDS COPYRIGHT 1998 DERWENT INFORMATION LTD
AN 84-154226 [25] WPIDS
DNC C84-064936
TI Compsn. contg. progestational agent and folic acid or deriv. - for

reducing hair loss in men.
DC B01
PA (MORT-I) MORTIMER C H
CYC 1
PI GB 2131292 A 840620 (8425)* 6 pp
GB 2131292 B 870311 (8710)
ADT GB 2131292 A GB 82-34226 821201
PRAI GB 82-34226 821201
IC **A61K007-06; A61K037-38**
AB GB 2131292 A UPAB: 930925
Pharmaceutical formulation which provides a progestationally active agent (I) and folic acid or a suitable deriv. of folic acid (II) is new.
Pref. (I) is medroxyprogesterone or its derivatives, esp. the acetate, other suitable cpds. being allylestrenol, gestronol hexanoate, norgestrel, norethisterone and hydroxy-progesterone hexanoate.
The formulation is used to **reduce** hair loss in men and even to provide an **increase** in **hair growth**. It lowers the level of plasma **dihydrotestosterone** without excessively lowering the plasma **testosterone** level, and therefore allows sexual potency and spermatogenesis to be substantially maintained while allowing the hair follicles to remain active and healthy.
0/2
FS CPI
FA AB
MC CPI: B01-C03; B01-C04; B01-C05; B01-C06; B06-D09; B12-G04; B12-L05

L130 ANSWER 87 OF 97 MEDLINE
AN 84275026 MEDLINE
DN 84275026
TI [Microsporum infection in a 3-month-old infant].
Microsporie chez un enfant de 3 mois.
AU Baudraz-Rosselet F; Ruffieux C; Grigoriu D
SO THERAPEUTISCHE UMSCHAU, (1984 Jun) 41 (6) 403-5.
Journal code: VPT. ISSN: 0040-5930.
CY Switzerland
DT Journal; Article; (JOURNAL ARTICLE)
LA French
EM 198411
CT Check Tags: Case Report; Human
Antifungal Agents: TU, therapeutic use
English Abstract
Griseofulvin: TU, therapeutic use
Hair: MI, microbiology
Imidazoles: TU, therapeutic use
Infant
*Microsporum: IP, isolation & purification
*Tinea Capitis: DI, diagnosis
Tinea Capitis: DT, drug therapy
RN 126-07-8 (Griseofulvin); **65899-73-2 (tioconazole)**
CN 0 (Antifungal Agents); 0 (Imidazoles)

L130 ANSWER 88 OF 97 BIOSIS COPYRIGHT 1998 BIOSIS
AN 85:345854 BIOSIS
DN BA80:15846
TI ANTI-**ANDROGEN TREATMENT** OF HIRSUTE WOMEN A STUDY ON STRESS RESPONSES.
AU LUNDBERG U; HANSSON U; ENEROTH P; FRANKENHAEUSER M; HAGENFELDT K
CS DEP. PSYCHOL., UNIV. STOCKHOLM, S-106 91 STOCKHOLM, SWED.
SO J PSYCHOSOM OBSTET GYNAECOL 3 (2). 1984. 79-92. CODEN: JPOGDP
LA English
AB Fifteen hirsute women with oligomenorrhea were compared with

age-matched, healthy, normally menstruating women during rest and experimentally induced stress. Comparisons were made before, and after 6 wk and 10-12 mo. of **treatment** of the patients with cyproterone acetate (CPA) combined with ethinylestradiol (EE2). CPA **treatment** in the patients was associated with a marked **reduction** in **testosterone** ($P < 0.005$) and androstenedione ($P < 0.005$) levels and a weak but significant ($P < 0.01$) **reduction** in **hair growth** (Ferriman and Gallway Hirsutes Score). CPA **treatment** combined with EE2 **increased** heart rate ($P < 0.02$) without any change in catecholamine excretion and was also associated with a considerable **increase** in plasma cortisol ($P < 0.0001$), probably due to an **increased** level of corticosteroid binding globulin (CBG). Differences in the correlational pattern for steroid hormones in the patients and the control subjects suggest an imbalance in adrenal steroid biosynthesis in the patients, which was normalized after CPA **treatment**. No changes in personality characteristics were noted after 1 yr of **treatment**.

ST CYPROTERONE ACETATE ETHYNYLESTRADIOL HORMONE-DRUG OLIGOMENORRHEA STEROID PERSONALITY

RN 57-63-6 (ETHYNYLESTRADIOL)
427-51-0 (CYPROTERONE ACETATE)

CC Mathematical Biology and Statistical Methods 04500
Behavioral Biology-Human Behavior *07004
Clinical Biochemistry; General Methods and Applications 10006
Biochemical Studies-Proteins, Peptides and Amino Acids 10064
Biochemical Studies-Sterols and Steroids 10067
Pathology, General and Miscellaneous-Therapy *12512
Metabolism-Sterols and Steroids *13008
Metabolism-Proteins, Peptides and Amino Acids *13012
Reproductive System-Physiology and Biochemistry *16504
Reproductive System-Pathology *16506
Endocrine System-Adrenals *17004
Endocrine System-Neuroendocrinology *17020
Integumentary System-Pathology *18506
Nervous System-Physiology and Biochemistry *20504
Psychiatry-General; Medical Psychology and Sociology *21001
Psychiatry-Psychophysiology *21003
Pharmacology-Clinical Pharmacology *22005
Pharmacology-Endocrine System *22016
Pharmacology-Integumentary System, Dental and Oral Biology *22020

BC **Hominidae 86215**

L130 ANSWER 89 OF 97 WPIDS COPYRIGHT 1998 DERWENT INFORMATION LTD
AN 83-777273 [40] WPIDS
DNC C83-094621
TI Topical compsn. for **stimulating hair growth** - contg. oxindole, pref. in aq. ethanol vehicle.
DC B01 D21
IN TSUCHIYA, W
PA (SHIS) SHISEIDO CO LTD; (TAKE) TAKEDA CHEM IND LTD; (TSUC-I) TSUCHIYA W
CYC 11
PI BE 896213 A 830919 (8340)* 37 pp
DE 3309813 A 831013 (8342)
FR 2523440 A 830923 (8343)
JP 58162512 A 830927 (8344)
AU 8312573 A 830922 (8345)
NL 8300972 A 831017 (8345)
GB 2122081 A 840111 (8402)
JP 59059606 A 840405 (8420)
JP 59059607 A 840405 (8420)
GB 2122081 B 860403 (8614)

CH 657774 A 860930 (8642)
CA 1222460 A 870602 (8726)
JP 01012725 B 890302 (8913)
IT 1162843 B 870401 (8924)
DE 3309813 C 920702 (9227) 15 pp A61K031-565 <--
US 1551 H 960604 (9628) 7 pp A61K031-56 <--
ADT JP 58162512 A JP 82-166193 820924; GB 2122081 A GB 83-7346 830317; JP 59059606 A JP 82-166194 820924; JP 59059607 A JP 82-45103 820320; DE 3309813 C DE 83-3309813 830318; US 1551 H Cont of US 83-475924 830316, Cont of US 84-659870 841012, Cont of US 87-91769 870827, Cont of US 89-426525 891024, Cont of US 90-559416 900725, Cont of US 91-729861 910710, Cont of US 92-899593 920618, US 93-132487 931006
PRAI JP 82-45103 820320; JP 82-166193 820924; JP 82-166194 820924
IC ICM **A61K031-56; A61K031-565**
ICS **A61K007-06; A61K031-12**; C07C000-00; C07J001-00
AB BE 896213 A UPAB: 930925
Topical compsn. for **stimulating growth** of **hair** contains at most 2% (pref. 0.001-2 wt.%) oxendolone (I; 16beta-ethyl-17beta-hydroxy-4-oestren-3-one) in a suitable vehicle or support and, if necessary, other usual additives. Partic. the vehicle is an aq. soln. contg. at least 30 wt.% ethanol, and compsns. are formulated as a pommade or emulsion.
The compsn. has no undesirable side effects and such as a systemic hormonal activity. (I) **inhibits** both **testosterone**-5alpha **reductase** and attachment of 5alpha-**dihydrotestosterone** to protein receptors. (I) is already known for treatment of benign prostatic hypertrophy.
0/0
FS CPI
FA AB
MC CPI: B01-C05; B12-G01; B12-L05; D08-B03

L130 ANSWER 90 OF 97 BIOSIS COPYRIGHT 1998 BIOSIS
AN 83:310078 BIOSIS
DN BA76:67570
TI INDUCTION OF PUBERTY BY PROLONGED PULSATILE LHRH ADMINISTRATION.
AU DELEMARRE-VAN DE WAAL H A; SCHOEMAKER J
CS DEP. OF PEDIATRICS, ACADEMIC HOSP. VRIJI UNIV., AMSTERDAM, THE NETHERLANDS.
SO ACTA ENDOCRINOL 102 (4). 1983. 603-609. CODEN: ACENA7 ISSN: 0001-5598
LA English
AB Pubertal maturation was induced in a 17.7 year old hypogonadotropic boy by pulsatile LHRH treatment. LHRH was administered in 3 periods. During period one 20 .mu.g LHRH pulses were given i.v. 16 times per day for 10 wk; during period two 2 .mu.g LHRH pulses i.v. 16 times per day for 12 wk. During period three 2 .mu.g LHRH pulses 16 times per day were given s.c. for 13 wk. Treatment was interrupted for 6 wk between period 1 and 2. Rapid initiation of pubertal maturation was evidenced by an increase of penile length and testicular volume as well as by **growth** of pubic **hair**. After 21 wk of treatment spermatozoa were observed in the ejaculate. Gonadotropin levels increased from prepubertal values into the supranormal range in the beginning of period 1, spontaneously declining to normal adult levels. A rapid increment of testicular volume during period 1 was also evidence for overstimulation. During period 2 gonadotropin levels were in the normal range. **Testosterone** levels were normal during period 1 and 2, although higher during period 1. Evidently, pulsatile LHRH treatment with 2 .mu.g per pulse i.v. 16 times per day is an adequate and feasible way to induce puberty in hypogonadotropic males with an intact pituitary. Under pulsatile LHRH treatment spermatogenesis takes place more rapidly than during normal puberty. Testicular hormones exert a negative feedback action at the

pituitary in the LHRH treated hypogonadotropic male. The supranormal levels of LH and FSH during the 1st weeks of treatment may be caused by a delayed reaction of the testicles to gonadotropin **stimulation** rather than to an overdose of LHRH. No evidence was found of a direct **inhibitory** action of LHRH on testicular function.

ST CHILD HORMONE-DRUG FSH LUTEINIZING HORMONE TESTICULAR HORMONES HYPO GONADOTROPIC SPERMATOZOA PENILE LENGTH TESTICULAR VOLUME PUBIC **HAIR**

RN 9002-67-9 (LUTEINIZING HORMONE)
9002-68-0 (FSH)
9034-40-6 (LHRH)

CC Cytology and Cytochemistry-Human 02508
Clinical Biochemistry; General Methods and Applications *10006
Biochemical Studies-Proteins, Peptides and Amino Acids 10064
Biochemical Studies-Sterols and Steroids 10067
Biochemical Studies-Carbohydrates 10068
Biophysics-Biocybernetics 10515
Pathology, General and Miscellaneous-Therapy *12512
Cardiovascular System-General; Methods 14501
Reproductive System-Physiology and Biochemistry *16504
Reproductive System-Pathology *16506
Endocrine System-Gonads and Placenta *17006
Endocrine System-Pituitary *17014
Endocrine System-Neuroendocrinology *17020
Integumentary System-General; Methods 18501
Integumentary System-Physiology and Biochemistry *18504
Pharmacology-Clinical Pharmacology 22005
Pharmacology-Endocrine System *22016
Pharmacology-Reproductive System; Implantation Studies *22028
Routes of Immunization, Infection and Therapy 22100
Pediatrics *25000

BC Hominidae 86215

L130 ANSWER 91 OF 97 BIOSIS COPYRIGHT 1998 BIOSIS

AN 84:242932 BIOSIS

DN BA77:75916

TI THE **STIMULATION** OF **HAIR GROWTH** IN THE FLANK ORGANS OF FEMALE HAMSTERS BY SUB CUTANEOUS **TESTOSTERONE** PROPIONATE AND ITS **INHIBITION** BY TOPICAL CYPROTERONE ACETATE DOSE RESPONSE STUDIES.

AU KASZYNSKI E

CS BIOLOGICAL SCI. DEP., GILLETTE RES. INST., 1413 RESEARCH BOULEVARD, ROCKVILLE, MD 20850, USA.

SO BR J DERMATOL 109 (5). 1983. 565-570. CODEN: BJDEAZ ISSN: 0007-0963

LA English

AB A dose-dependent increase in the mass of flank organ **hair** was produced in 11-wk-old female hamsters by s.c. injected **testosterone** propionate. The mass of **androgen-stimulated** flank organ **hair** was decreased bilaterally in a dose-dependent manner by cyproterone acetate applied topically to 1 flank organ of each hamster.

ST HORMONE-DRUG METABOLIC-DRUG

RN 57-85-2 (TESTOSTERONE PROPIONATE)
427-51-0 (CYPROTERONE ACETATE)

CC Biochemical Methods-Sterols and Steroids 10057
Biochemical Studies-Sterols and Steroids 10067
Chordate Body Regions-Extremities 11318
Endocrine System-Gonads and Placenta *17006
Integumentary System-General; Methods *18501
Integumentary System-Physiology and Biochemistry *18504
Pharmacology-Drug Metabolism; Metabolic Stimulators *22003
Pharmacology-Endocrine System *22016

Pharmacology-Integumentary System, Dental and Oral Biology
***22020**
Routes of Immunization, Infection and Therapy 22100
BC Cricetidae 86310

L130 ANSWER 92 OF 97 MEDLINE
AN 84057123 MEDLINE
DN 84057123
TI Hair copper and zinc concentrations in handicapped children treated with anticonvulsants.
AU Ikeda T; Higashi A; Matsukura M; Matsuda I
SO DEVELOPMENTAL PHARMACOLOGY AND THERAPEUTICS, (1983) 6 (6) 381-7. Journal code: EAF. ISSN: 0379-8305.
CY Switzerland
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198403
AB Hair copper and zinc contents were measured in 95 handicapped children aged from 4 to 17 years and 48 age- and sex-matched control children. The patients consisted of 5 groups: children untreated with anticonvulsants (n = 7), those treated with phenytoin and phenobarbital (n = 32), those treated with phenytoin, phenobarbital and diazepam (n = 18), those treated with diazepam alone (n = 16) and those treated with phenobarbital alone (n = 12). The patients were all institutionalized in the same medical care unit and received the same diet, containing decreased amounts of copper (75% of control) and sufficient amounts of zinc. The patients belonging to all of the 5 groups had less amounts of hair copper (p less than 0.05) and erythrocyte hemoglobin (p less than 0.01) in comparison to controls. The patients receiving diazepam alone or in addition to other anticonvulsants had significantly less hair zinc content (p less than 0.05) in comparison to controls or other patient groups. Thus, diazepam seemed to have an adverse effect, producing zinc deficiency.
CT Check Tags: Female; Human; Male; Support, Non-U.S. Gov't
Adolescence
*Anticonvulsants: AE, adverse effects
Child
Child, Preschool
*Copper: ME, metabolism
Diazepam: AE, adverse effects
Disabled Persons
***Hair: AN, analysis**
Hemoglobins: ME, metabolism
Phenobarbital: AE, adverse effects
Phenytoin: AE, adverse effects
*Zinc: ME, metabolism
RN 439-14-5 (Diazepam); **50-06-6 (Phenobarbital)**; 57-41-0 (Phenytoin); 7440-50-8 (Copper); 7440-66-6 (Zinc)
CN 0 (Anticonvulsants)

L130 ANSWER 93 OF 97 BIOSIS COPYRIGHT 1998 BIOSIS
AN 83:170888 BIOSIS
DN BA75:20888
TI NORMALIZATION OF **TESTOSTERONE** LEVELS USING A LOW ESTROGEN CONTAINING ORAL CONTRACEPTIVE IN WOMEN WITH POLY CYSTIC OVARY SYNDROME.
AU RAJ S G; RAJ M H G; TALBERT L M; SLOAN C S; HICKS B
CS DEP. OBSTETRICS GYNECOL., UNIV. NORTH CAROLINA SCH. MED., CHAPEL HILL, NORTH CAROLINA.
SO OBSTET GYNECOL 60 (1). 1982. 15-19. CODEN: OBGNAS ISSN: 0029-7844
LA English
AB Oral contraceptives **reduce** the elevated **androgen**

levels in polycystic ovary disease. **Treatment** with oral contraceptives is associated with undesirable side effects because of their high estrogen content. The effects of low estrogen-containing oral contraceptive (Loestrin:norethindrone acetate 1.5 mg and ethinyl estradiol 30 .mu.g) were studied on 25 women with polycystic ovary disease of ovarian origin. Loestrin **treatment** normalized the elevated luteinizing hormone and total and unbound **testosterone** levels and **increased** **testosterone** binding globulin levels. In a time-course study, unbound **testosterone** declined within a week of initiating **treatment** and by 12-16 wk was completely normal. **Reduction** in **hair growth** and improvement in complexion were noted by .apprx. 12-16 wk. Side effects of **treatment** were minimal.

ST HUMAN LOESTRIN NORETHINDRONE ACETATE ETHYNYL ESTRADIOL HORMONE-DRUG LUTEINIZING HORMONE **HAIR GROWTH** COMPLEXION PHARMACODYNAMICS

RN 57-63-6 (ETHYNYL ESTRADIOL)
58-22-0 (TESTOSTERONE)
9002-67-9 (LUTEINIZING HORMONE)

CC Cytology and Cytochemistry-Human 02508
Clinical Biochemistry; General Methods and Applications 10006
Biochemical Studies-Proteins, Peptides and Amino Acids 10064
Biochemical Studies-Sterols and Steroids 10067
Biochemical Studies-Carbohydrates 10068
Pathology, General and Miscellaneous-Therapy 12512
Metabolism-Carbohydrates 13004
Metabolism-Sterols and Steroids 13008
Metabolism-Proteins, Peptides and Amino Acids 13012
Blood, Blood-Forming Organs and Body Fluids-Blood and Lymph Studies 15002
Reproductive System-Pathology *16506
Endocrine System-Gonads and Placenta *17006
Endocrine System-Pituitary *17014
Integumentary System-Pathology 18506
Dental and Oral Biology-General; Methods 19001
Pharmacology-Drug Metabolism; Metabolic Stimulators 22003
Pharmacology-Clinical Pharmacology 22005
Pharmacology-Endocrine System *22016
Pharmacology-Integumentary System, Dental and Oral Biology 22020
Pharmacology-Reproductive System; Implantation Studies *22028
Routes of Immunization, Infection and Therapy 22100
Toxicology-Pharmacological Toxicology *22504

BC **Hominidae 86215**

L130 ANSWER 94 OF 97 MEDLINE
AN 81241457 MEDLINE
DN 81241457
TI Detection of phenobarbital in bloodstains, semen, seminal stains, saliva, saliva stains, perspiration stains, and hair.
AU Smith F P; Pomposini D A
SO JOURNAL OF FORENSIC SCIENCES, (1981 Jul) 26 (3) 582-6.
Journal code: I5Z. ISSN: 0022-1198.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198111
CT Check Tags: Human; Male; Support, Non-U.S. Gov't
Blood Stains
Forensic Medicine
Hair: AN, analysis

*Phenobarbital: AN, analysis
Phenobarbital: BL, blood
Radioimmunoassay
Saliva: AN, analysis
Semen: AN, analysis
RN **50-06-6 (Phenobarbital)**

L130 ANSWER 95 OF 97 BIOSIS COPYRIGHT 1998 BIOSIS
AN 81:234432 BIOSIS
DN BA72:19416
TI DIFFERENTIAL EFFECT OF 13-CIS RETINOIC-ACID AND AN AROMATIC RETINOID RO-10-9359 ON THE SEBACEOUS GLANDS OF THE HAMSTER FLANK ORGAN.
AU GOMERZ E C
CS DEP. DERMATOL., UNIV. CALIF., DAVIS, UCD, PROFESSIONAL BUILD., 4301 X. ST., SACRAMENTO, CALIF., 95817.
SO J INVEST DERMATOL 76 (1). 1981. 68-69. CODEN: JIDEAE ISSN: 0022-202X
LA English
AB The effect of s.c. administered 13-cis-retinoic acid and an aromatic retinoid (Ro 10-9359 [3,7-dimethyl-9-(2,5,6-trimethyl-4-methoxyphenyl)-2,4,6,8-trans-nonatetraenoic acid ethyl ester]) on the sebaceous glands of the hamster flank organ were compared. 13-cis-Retinoic acid caused a marked diminution of sebaceous gland size without affecting other **androgen**-dependent structures. The aromatic retinoid derivative showed no effect upon any of the flank organ components. Studies using **androgen-stimulated** female confirmed the previous finding that 13-cis-retinoic acid prevented the **growth** of sebaceous glands without affecting the development of dermal pigmentation, or large pigmented **hair** follicles. The aromatic retinoid derivative showed slight, if any, effect upon sebaceous gland size, and no effect upon pigmentation or pigmented follicle development. The findings with this model system suggest that any efficacy of Ro 10-9359 in the treatment of acne would be by some mode other than the **inhibition** of sebum production.
ST MODEL SEBUM **HAIR** FOLLICLE 3 7 DI METHYL-9-2 5 6-TRIMETHYL-4-METHOXYPHENYL-2 4 6 8-TRANS NONA TETRAENOIC-ACID ETHYL ESTER METABOLIC-DRUG DERMATOLOGICAL-DRUG **ANDROGEN** PHARMACODYNAMICS ACNE
RN 4759-48-2 (13-CIS RETINOIC-ACID)
54350-48-0 (RO-10-9359)
CC Biochemical Studies-Vitamins 10063
Biochemical Studies-Lipids 10066
Biophysics-Molecular Properties and Macromolecules 10506
Biophysics-Biocybernetics 10515
Pathology, General and Miscellaneous-Inflammation and Inflammatory Disease 12508
Metabolism-Lipids 13006
Metabolism-Fat-Soluble Vitamins *13016
Blood, Blood-Forming Organs and Body Fluids-Other Body Fluids *15010
Integumentary System-General; Methods 18501
Integumentary System-Pathology *18506
Pharmacology-Drug Metabolism; Metabolic Stimulators *22003
Pharmacology-Integumentary System, Dental and Oral Biology *22020
Routes of Immunization, Infection and Therapy 22100
BC Cricetidae 86310

L130 ANSWER 96 OF 97 BIOSIS COPYRIGHT 1998 BIOSIS
AN 81:212908 BIOSIS
DN BA71:82900
TI INDUCTION OF PUBERTY IN BOYS WITH DELAYED ADOLESCENCE BY METHANDROSTENOLONE.
AU DICKERMAN Z; SHUPER A; PRAGER-LEWIN R; LAHMY O; LARON Z
CS INSTITUTE OF PEDIATRIC AND ADOLESCENT ENDOCRINOLOGY, BEILINSON

MEDICAL CENTRE, PETAH TIKVA, ISRAEL.
SO EUR J PEDIATR 135 (1). 1980 (RECD. 1981). 59-64. CODEN: EJPEDT ISSN: 0340-6199
LA English
AB Methandrostenolone administration at a daily dose of 0.03 mg/kg for 3 mo. was successful inducing puberty in 9 boys (aged 14 1/2 .+-. 1/2 yr, m [mean] .+-. SD) with delayed puberty and studied in the prepubertal stage. At 1 yr after initiation of treatment they reached a mid-pubertal stage (testicular volume 6 .+-. 2 ml and pubic **hair** development Tanner stage 3-4). At the same time **growth** velocity accelerated from 5.3 .+-. 1.5 to 8.5 .+-. 3.4 cm/yr and bone age advanced from 10 3/4 .+-. 3/4 to 13 .+-. 1/2 yr (m .+-. SD). During treatment there was suppression of basal plasma LH [lutropin] and FSH [follitropin] (m .+-. SD) from 1.3 .+-. 0.3 to 0.5 .+-. 0.2 mIU/ml ($P < 0.001$) and from 1.4 .+-. 0.8 to 0.8 .+-. 0.3 mIU/ml ($P < 0.05$), respectively, and of the LH response to LRH [luliberin] (50 .mu.g/m2 i.v.) from 5.2 .+-. 1.0 to 1.9 .+-. 0.6 mIU/ml ($P < 0.001$). After discontinuation of methandrostenolone there was a significant and prolonged elevation of the basal plasma LH (2.0 .+-. 0.4 mIU/ml) and **testosterone** levels (from 24 .+-. 7.7 to 175.6 .+-. 67.5 ng/dl, $P < 0.01$) and an enhanced LH response to LRH (8.3 .+-. 2.4 mIU/ml, $P < 0.05$), compared to the pretreatment levels. Eleven prepubertal boys with constitutional short stature (aged 9 1/4 .+-. 3/4 yr, m .+-. SD) maintained their prepubertal state 1 yr following the same therapeutic regime with methandrostenolone. No significant changes in the basal plasma **testosterone** and gonadotropin levels, or the responses to LRH, were noted in this group. During treatment a significant increase in **growth** velocity was noted (from 4.1 .+-. 1.7 to 9.7 .+-. 3.0 cm/yr, $P < 0.02$), with a subsequent decrease to 5.4 .+-. 2.9 cm/yr (m .+-. SD) which was not significantly different from the pretreatment value. Bone age advanced from 6 1/4 .+-. 1 before treatment to 8 .+-. 1 1/2 yr 12 mo. following methandrostenolone administration. Apparently, methandrostenolone can induce puberty in boys with delayed puberty if administered in the prepubertal stage, but not in younger prepubertal boys with short stature. The concomitant changes in the basal plasma **testosterone** and gonadotropin levels, and their response to LRH stimulation, which were found in the boys with delayed puberty, indicate that a certain degree of maturation of the hypothalamic pituitary gonadal axis is probably needed to permit induction of puberty by methandrostenolone. The effect of this drug is due in part to its **androgenic** potency and probably also to its **modulation** of negative feedback in the hypothalamic-pituitary-gonadal axis, causing a rebound phenomenon following brief suppression.
ST HUMAN LULIBERIN HORMONE-DRUG DIAGNOSTIC-DRUG LUTROPIN FOLLITROPIN **TESTOSTERONE** BONE AGE
RN 58-22-0 (TESTOSTERONE)
72-63-9 (METHANDROSTENOLONE)
9002-67-9 (LUTROPIN)
9002-68-0 (FOLLITROPIN)
9034-40-6 (LULIBERIN)
CC Mathematical Biology and Statistical Methods 04500
Clinical Biochemistry; General Methods and Applications *10006
Biochemical Studies-Proteins, Peptides and Amino Acids 10064
Biochemical Studies-Sterols and Steroids 10067
Biochemical Studies-Carbohydrates 10068
Biophysics-Biocybernetics 10515
Pathology, General and Miscellaneous-Diagnostic 12504
Pathology, General and Miscellaneous-Therapy *12512
Metabolism-Carbohydrates 13004
Metabolism-Sterols and Steroids *13008
Metabolism-Proteins, Peptides and Amino Acids *13012
Cardiovascular System-General; Methods 14501

Blood, Blood-Forming Organs and Body Fluids-Blood and Lymph Studies 15002
Reproductive System-Physiology and Biochemistry *16504
Endocrine System-Gonads and Placenta *17006
Endocrine System-Pituitary *17014
Endocrine System-Neuroendocrinology *17020
Bones, Joints, Fasciae, Connective and Adipose Tissue-Physiology and Biochemistry *18004
Integumentary System-Physiology and Biochemistry 18504
Nervous System-Physiology and Biochemistry 20504
Pharmacology-Drug Metabolism; Metabolic Stimulators 22003
Pharmacology-Clinical Pharmacology 22005
Pharmacology-Connective Tissue, Bone and Collagen-Acting Drugs 22012
Pharmacology-Endocrine System *22016
Pharmacology-Reproductive System; Implantation Studies *22028
Routes of Immunization, Infection and Therapy 22100
Pediatrics 25000
Developmental Biology-Embryology-Morphogenesis, General 25508
BC Hominidae 86215

L130 ANSWER 97 OF 97 BIOSIS COPYRIGHT 1998 BIOSIS
AN 78:173738 BIOSIS
DN BA65:60738
TI EFFECT OF CYPROTERONE ACETATE ON **HAIR GROWTH** SEBACEOUS SECRETION AND ENDOCRINE PARAMETERS IN A HIRSUTE SUBJECT.
AU EBLING F J; THOMAS A K; COOKE I D; RANDALL V A; SKINNER J; CAWOOD M
CS DEP. ZOOL., UNIV., SHEFFIELD S10 2TN, YORKS., ENGL., UK.
SO BR J DERMATOL 97 (4). 1977 371-382. CODEN: BJDEAZ ISSN: 0007-0963
LA English
AB The quantitative changes in body **hair growth** and sebaceous secretion, as well as plasma sex hormone binding globulin, luteinizing hormone, follicle stimulating hormone, **testosterone** and androstenedione were measured in a hirsute woman aged 21 yr under reverse sequential **treatment** with cyproterone acetate and ethinyl estradiol. The subject before **treatment** had normal excretion of 17-oxosteroids, 17-oxogenic steroids, androsterone, dehydroepiandrosterone and etiocholanolone. The rate of **hair growth** on the thigh and the average **hair** diameter was significantly **reduced** after only 2 **treatment** cycles. After 6-7 cycles the length attained by the terminal hairs was **reduced** and this appeared to be due mainly to change in **growth** rate rather than to alteration in the duration of anagen. The shorter and thinner hairs also had a much shorter region of pigmented medulla. A progressive decrease in the extent and continuity of the medulla could be detected after 3 cycles of **treatment**. Sebaceous secretion was also **reduced** after 2 **treatment** cycles. Steady improvement of the pustular acne occurred thereafter. Sex hormone binding globulin levels were low before **treatment**, unaltered by a first cycle of cyproterone acetate alone, but **increased** by addition of ethinyl estradiol. Gonadotrophins remained low throughout, while **testosterone** and androstenedione levels, initially high, were substantially suppressed.
ST HUMAN ETHYNYL ESTRADIOL HORMONE-DRUG DERMATOL-DRUGS LUTEINIZING HORMONE FOLLICLE STIMULATING HORMONE **TESTOSTERONE** ANDROSTENEDIONE SEX HORMONE BINDING GLOBULIN PUSTULAR ACNE
RN 57-63-6 (ETHYNYL ESTRADIOL)
58-22-0 (TESTOSTERONE)
63-05-8 (ANDROSTENEDIONE)
427-51-0 (CYPROTERONE ACETATE)
CC Clinical Biochemistry; General Methods and Applications 10006

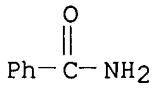
Biochemical Studies-Proteins, Peptides and Amino Acids 10064
Biochemical Studies-Sterols and Steroids 10067
Biochemical Studies-Carbohydrates 10068
Pathology, General and Miscellaneous-Inflammation and Inflammatory Disease 12508
Pathology, General and Miscellaneous-Therapy 12512
Metabolism-Carbohydrates *13004
Metabolism-Sterols and Steroids *13008
Metabolism-Proteins, Peptides and Amino Acids *13012
Blood, Blood-Forming Organs and Body Fluids-Blood and Lymph Studies 15002
Endocrine System-Gonads and Placenta *17006
Endocrine System-Pituitary *17014
Integumentary System-Physiology and Biochemistry 18504
Integumentary System-Pathology *18506
Pharmacology-Drug Metabolism; Metabolic Stimulators *22003
Pharmacology-Clinical Pharmacology 22005
Pharmacology-Endocrine System *22016
Pharmacology-Integumentary System, Dental and Oral Biology *22020

BC **Hominidae 86215**

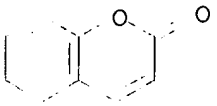
=> d bib abs hitstr

L5 ANSWER 1 OF 4 HCAPLUS COPYRIGHT 1998 ACS
AN 1998:484914 HCAPLUS
DN 129:140464
TI Reduction of hair growth by an inhibitor of a DNA topoisomerase
IN **Styczynski, Peter; Ahluwalia, Gurpreet S.**
PA Handelman, Joseph, H., USA
SO PCT Int. Appl., 16 pp.
CODEN: PIXXD2
PI WO 9829086 A1 19980709
DS W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG
AI WO 97-US24268 19971223
PRAI US 96-777803 19961231
DT Patent
LA English
AB Mammalian hair growth is reduced by applying to the skin an inhibitor of a DNA topoisomerase. Application of a soln. of 10% nalidixic acid in 70% ethanol and 30% propylene glycol inhibited hair growth in hamster by 63%.
IT **80449-01-0**, DNA topoisomerase
RL: BSU (Biological study, unclassified); BIOL (Biological study) (inhibitors; redn. of hair growth by inhibitor of DNA topoisomerase)
RN 80449-01-0 HCAPLUS
CN Isomerase, deoxyribonucleate topo- (9CI) (CA INDEX NAME)

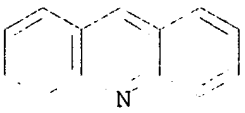
*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
IT **55-21-0**, Benzamide **91-64-5D**, Coumarin, derivs.
260-94-6, Acridine **303-81-1**, Novobiocin
389-08-2, Nalidixic acid **465-21-4**, Bufalin
476-66-4, Ellagic acid **519-23-3**, Ellipticine
1402-38-6, Actinomycin **4375-07-9**,
Epipodophyllotoxin **4375-07-9D**, Epipodophyllotoxin, derivs.
16502-01-5D, 1,2,3,4-Tetrahydro-.beta.-carboline, derivs.
20342-64-7D, 1H-Indole-4,7-dione, derivs. **21416-67-1**
24584-09-6, Dexrazoxane **29767-20-2**, Teniposide
33419-42-0, Etoposide **37045-16-2**,
3-Benzylquinoline **51264-14-3**, Amsacrine **52259-65-1**
, FAgaronine **69408-81-7**, Amonafide **97534-21-9**,
Merbarone **100440-25-3**, Terpentecin **108121-76-2**,
Anthracenedione **123577-49-1** **129564-92-7**,
Azatoxin **131190-63-1**, Saintopin **142805-56-9**,
Topoisomerase II **143180-75-0** **146555-80-8**,
Makaluvamine C **158734-24-8**, Dehydrokuanoniamine b
158758-41-9, Shermilamine C **163564-63-4**, Elenic
acid **210095-61-7D**, 4-substituted derivs.
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(redn. of hair growth by inhibitor of DNA topoisomerase)
RN 55-21-0 HCAPLUS
CN Benzamide (8CI, 9CI) (CA INDEX NAME)



RN 91-64-5 HCAPLUS
CN 2H-1-Benzopyran-2-one (9CI) (CA INDEX NAME)

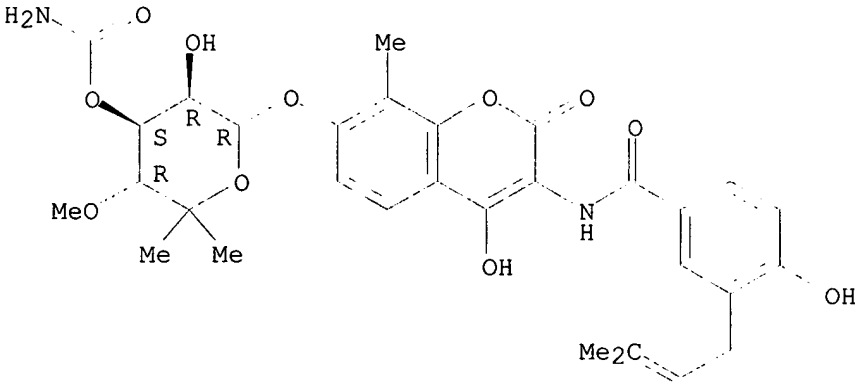


RN 260-94-6 HCAPLUS
CN Acridine (8CI, 9CI) (CA INDEX NAME)

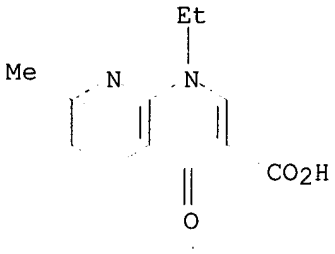


RN 303-81-1 HCAPLUS
CN Benzamide, N-[7-[[3-O-(aminocarbonyl)-6-deoxy-5-C-methyl-4-O-methyl-.alpha.-L-lyxo-hexopyranosyl]oxy]-4-hydroxy-8-methyl-2-oxo-2H-1-benzopyran-3-yl]-4-hydroxy-3-(3-methyl-2-butenyl)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.

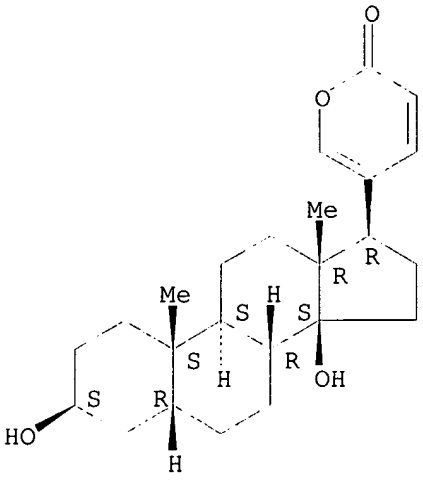


RN 389-08-2 HCAPLUS
CN 1,8-Naphthyridine-3-carboxylic acid, 1-ethyl-1,4-dihydro-7-methyl-4-oxo- (8CI, 9CI) (CA INDEX NAME)

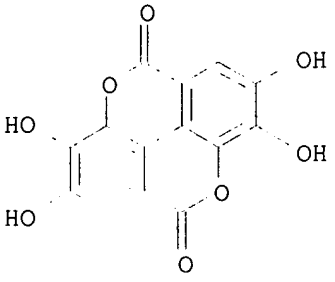


RN 465-21-4 HCAPLUS
CN Bufa-20,22-dienolide, 3,14-dihydroxy-, (3.beta.,5.beta.)- (9CI) (CA INDEX NAME)

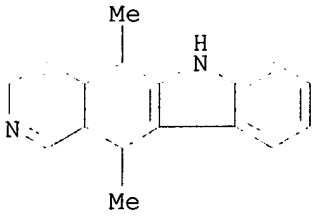
Absolute stereochemistry.



RN 476-66-4 HCAPLUS
CN [1]Benzopyrano[5,4,3-cde][1]benzopyran-5,10-dione, 2,3,7,8-tetrahydroxy- (7CI, 8CI, 9CI) (CA INDEX NAME)



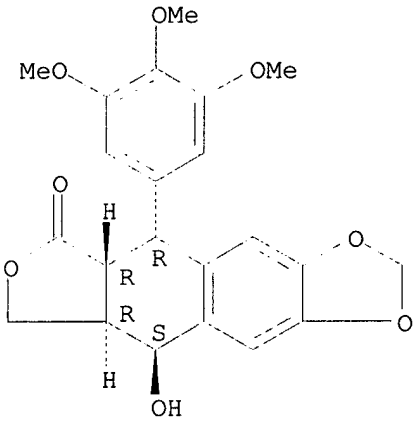
RN 519-23-3 HCAPLUS
CN 6H-Pyrido[4,3-b]carbazole, 5,11-dimethyl- (7CI, 8CI, 9CI) (CA INDEX NAME)



RN 1402-38-6 HCAPLUS
CN Actinomycin (9CI) (CA INDEX NAME)

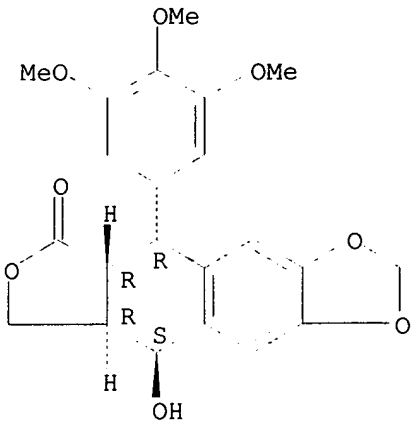
*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 4375-07-9 HCAPLUS
CN Furo[3',4':6,7]naphtho[2,3-d]-1,3-dioxol-6(5aH)-one,
5,8,8a,9-tetrahydro-9-hydroxy-5-(3,4,5-trimethoxyphenyl)-,
(5R,5aR,8aR,9S)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.

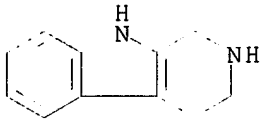


RN 4375-07-9 HCAPLUS
CN Furo[3',4':6,7]naphtho[2,3-d]-1,3-dioxol-6(5aH)-one,
5,8,8a,9-tetrahydro-9-hydroxy-5-(3,4,5-trimethoxyphenyl)-,
(5R,5aR,8aR,9S)- (9CI) (CA INDEX NAME)

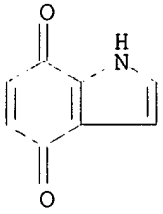
Absolute stereochemistry.



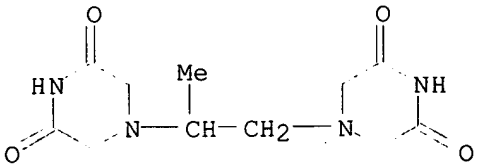
RN 16502-01-5 HCAPLUS
CN 1H-Pyrido[3,4-b]indole, 2,3,4,9-tetrahydro- (6CI, 8CI, 9CI) (CA INDEX NAME)



RN 20342-64-7 HCAPLUS
CN 1H-Indole-4,7-dione (9CI) (CA INDEX NAME)



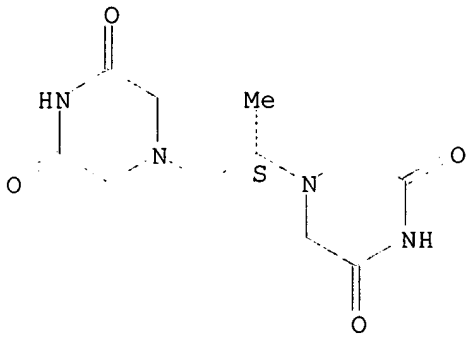
RN 21416-67-1 HCAPLUS
CN 2,6-Piperazinedione, 4,4'-(1-methyl-1,2-ethanediyl)bis- (9CI) (CA INDEX NAME)



RN 24584-09-6 HCAPLUS
CN 2,6-Piperazinedione, 4,4'-[(1S)-1-methyl-1,2-ethanediyl]bis- (9CI)

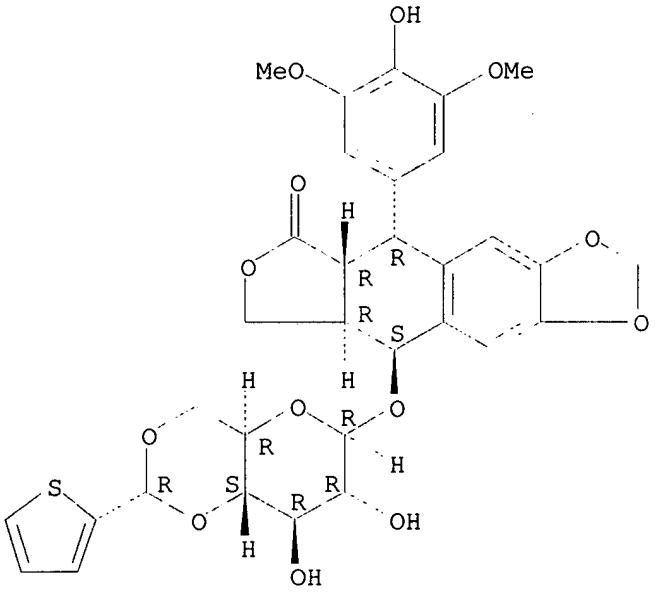
(CA INDEX NAME)

Absolute stereochemistry.



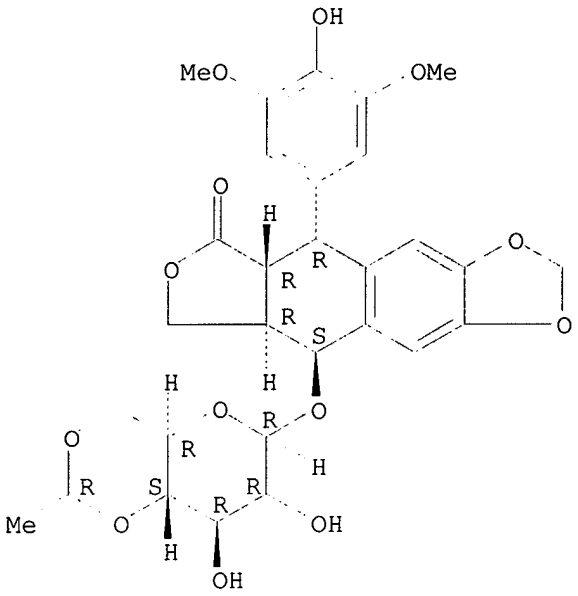
RN 29767-20-2 HCAPLUS
CN Furo[3',4':6,7]naphtho[2,3-d]-1,3-dioxol-6(5aH)-one, 5,8,8a,9-tetrahydro-5-(4-hydroxy-3,5-dimethoxyphenyl)-9-[[4,6-O-[(R)-2-thienylmethylene]-.beta.-D-glucopyranosyl]oxy]-, (5R,5aR,8aR,9S)-(9CI) (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).

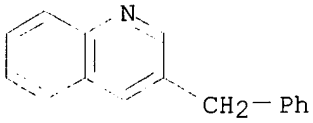


RN 33419-42-0 HCAPLUS
CN Furo[3',4':6,7]naphtho[2,3-d]-1,3-dioxol-6(5aH)-one, 9-[[4,6-O-(1R)-ethylidene-.beta.-D-glucopyranosyl]oxy]-5,8,8a,9-tetrahydro-5-(4-hydroxy-3,5-dimethoxyphenyl)-, (5R,5aR,8aR,9S)-(9CI) (CA INDEX NAME)

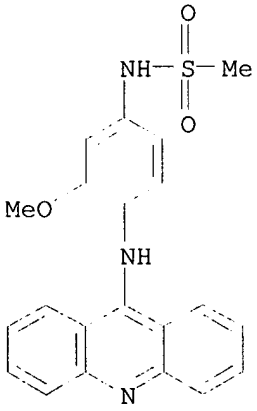
Absolute stereochemistry.



RN 37045-16-2 HCAPLUS
CN Quinoline, 3-(phenylmethyl)- (9CI) (CA INDEX NAME)

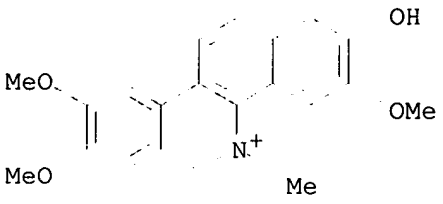


RN 51264-14-3 HCAPLUS
CN Methanesulfonamide, N-[4-(9-acridinylamino)-3-methoxyphenyl]- (9CI) (CA INDEX NAME)

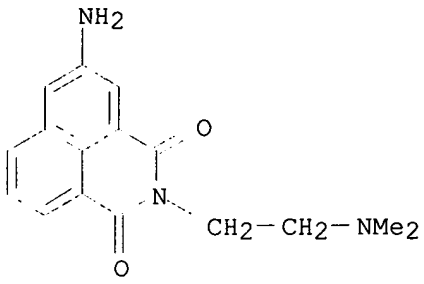


RN 52259-65-1 HCAPLUS
CN Benzo[c]phenanthridinium, 2-hydroxy-3,8,9-trimethoxy-5-methyl- (9CI)

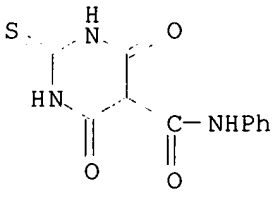
(CA INDEX NAME)



RN 69408-81-7 HCAPLUS
CN 1H-Benz[de]isoquinoline-1,3(2H)-dione, 5-amino-2-[2-(dimethylamino)ethyl]- (9CI) (CA INDEX NAME)

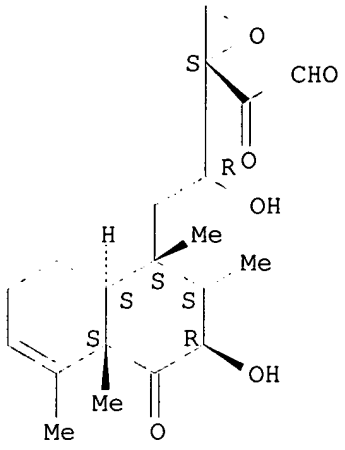


RN 97534-21-9 HCAPLUS
CN 5-Pyrimidinecarboxamide, hexahydro-4,6-dioxo-N-phenyl-2-thioxo- (9CI) (CA INDEX NAME)



RN 100440-25-3 HCAPLUS
CN Oxiraneacetaldehyde, 2-[(1S)-1-hydroxy-2-[(1R,2R,3S,4aR,8aR)-1,2,3,4,4a,7,8,8a-octahydro-3-hydroxy-1,2,4a,5-tetramethyl-4-oxo-1-naphthalenyl]ethyl]-.alpha.-oxo-, (2R)-rel-(-)- (9CI) (CA INDEX NAME)

Rotation (-). Absolute stereochemistry unknown.



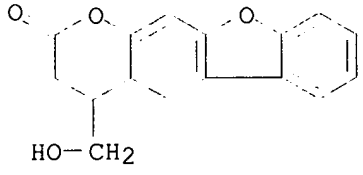
RN 108121-76-2 HCAPLUS
CN Anthracenedione (9CI) (CA INDEX NAME)

CM 1

CRN 96879-01-5
CMF C14 H20 O2
CCI IDS
CDES 8:ID

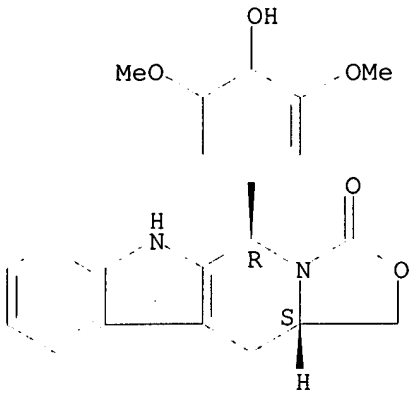
2 (D2═O)

RN 123577-49-1 HCAPLUS
CN 2H-Benzofuro[3,2-g]-1-benzopyran-2-one, 4-(hydroxymethyl)- (9CI) (CA INDEX NAME)

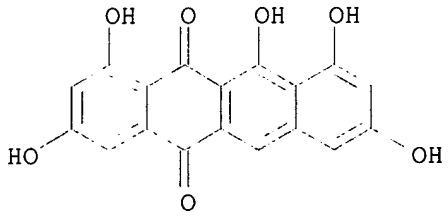


RN 129564-92-7 HCAPLUS
CN 1H,3H-Oxazolo[3',4':1,6]pyrido[3,4-b]indol-3-one, 5,6,11,11a-tetrahydro-5-(4-hydroxy-3,5-dimethoxyphenyl)-, (5R,11aS)-(9CI) (CA INDEX NAME)

Absolute stereochemistry.



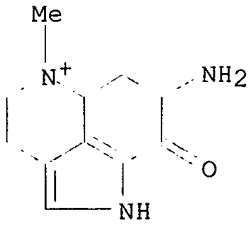
RN 131190-63-1 HCAPLUS
CN 5,12-Naphthacenedione, 1,3,8,10,11-pentahydroxy- (9CI) (CA INDEX NAME)



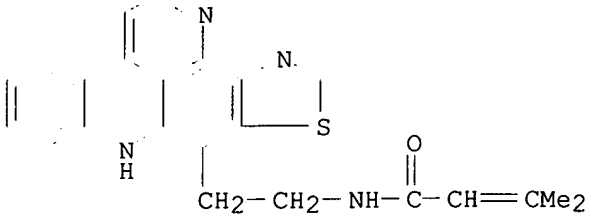
RN 142805-56-9 HCAPLUS
CN Isomerase, deoxyribonucleate topo-, II (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 143180-75-0 HCAPLUS
CN Isomerase, deoxyribonucleate topo-, I (9CI) (CA INDEX NAME)

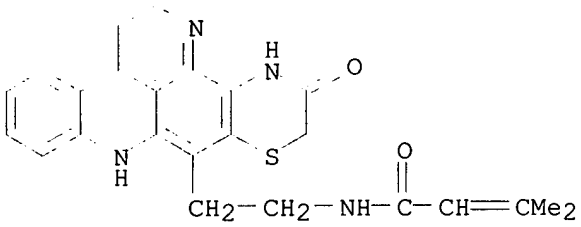
*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 146555-80-8 HCAPLUS
CN Pyrrolo[4,3,2-de]quinolinium, 7-amino-1,3,4,8-tetrahydro-5-methyl-8-oxo- (9CI) (CA INDEX NAME)



RN 158734-24-8 HCAPLUS
CN 2-Butenamide, 3-methyl-N-[2-(8H-pyrido[4,3,2-mn]thiazolo[4,5-b]acridin-9-yl)ethyl]- (9CI) (CA INDEX NAME)

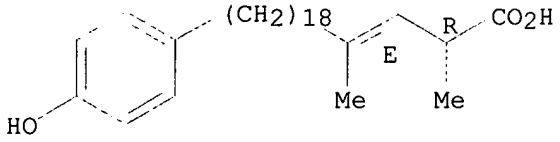


RN 158758-41-9 HCAPLUS
CN 2-Butenamide, 3-methyl-N-[2-(8,11,12,13-tetrahydro-12-oxopyrido[4,3,2-mn][1,4]thiazino[3,2-b]acridin-9-yl)ethyl]- (9CI) (CA INDEX NAME)

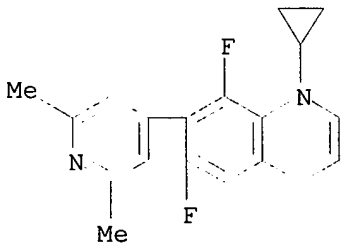


RN 163564-63-4 HCAPLUS
CN 3-Docosenoic acid, 22-(4-hydroxyphenyl)-2,4-dimethyl-, (2R,3E)- (9CI) (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).
Double bond geometry as shown.



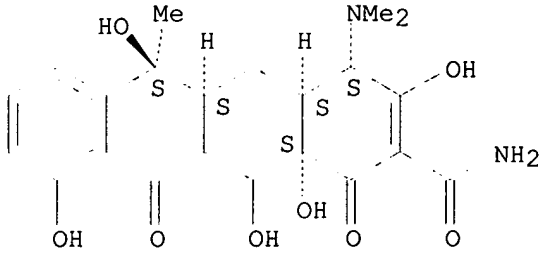
RN 210095-61-7 HCAPLUS
CN Quinoline, 1-cyclopropyl-7-(2,6-dimethyl-4-pyridinyl)-6,8-difluoro-1,4-dihydro- (9CI) (CA INDEX NAME)



=> d bib abs hitstr 2

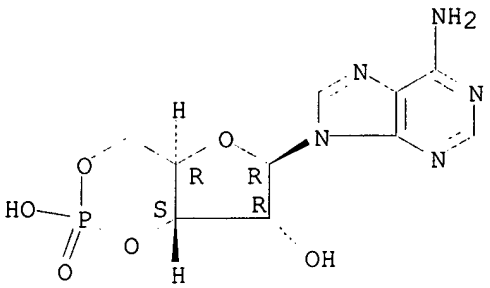
L5 ANSWER 2 OF 4 HCAPLUS COPYRIGHT 1998 ACS
AN 1998:402282 HCAPLUS
DN 129:71946
TI Reduction of hair growth
IN **Styczynski, Peter; Ahluwalia, Gurpreet S.;** Shander, Douglas
PA Handelman, Joseph, H., USA; Styczynski, Peter; Ahluwalia, Gurpreet S.; Shander, Douglas
SO PCT Int. Appl., 15 pp.
CODEN: PIXXD2
PI WO 9825580 A1 19980618
DS W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG
AI WO 97-US22587 19971212
PRAI US 96-764980 19961213
DT Patent
LA English
AB Mammalian hair growth is reduced by inhibiting the activity of a matrix metalloproteinase (MMP) in the skin. For example, bromo cAMP was dissolved in a vehicle contg. water 68, ethanol 16, propylene glycol 5, dipropylene glycol 5, benzyl alc. 4, and propylene carbonate 2 % to obtain a 10 % concn. When the compn. was tested by the Golden Syrian hamster assay, it provided .apprx.80 % redn. in hair growth.
IT **60-54-8**, Tetracycline **60-92-4D**, CAMP, bromo derivs. **66-71-7**, o-Phenanthroline **139-85-5**, Protocatechuic aldehyde **564-25-0**, Doxycycline **914-00-1**, Methacycline **2998-57-4**, Estramustine **10118-90-8**, Minocycline **13434-13-4**, Actinonin **25378-27-2**, Eicosapentaenoic acid **51036-13-6**, N-Chlorotaurine **130370-60-4**, Batimastat **140923-32-6**, Matlystatin B **141368-50-5** **153743-26-1** **154039-60-8**, Marimastat **157549-53-6** **209056-82-6**
RL: BAC (Biological activity or effector, except adverse); BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(matrix metalloproteinase inhibitors for redn. of unwanted hair growth)
RN 60-54-8 HCAPLUS
CN 2-Naphthacenecarboxamide, 4-(dimethylamino)-1,4,4a,5,5a,6,11,12a-octahydro-3,6,10,12,12a-pentahydroxy-6-methyl-1,11-dioxo-, (4S,4aS,5aS,6S,12aS)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.

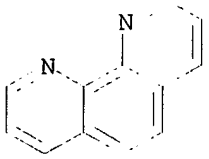


RN 60-92-4 HCAPLUS
CN Adenosine, cyclic 3',5'-(hydrogen phosphate) (8CI, 9CI) (CA INDEX NAME)

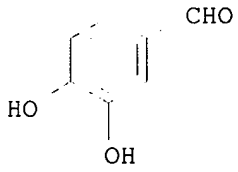
Absolute stereochemistry.



RN 66-71-7 HCAPLUS
CN 1,10-Phenanthroline (7CI, 8CI, 9CI) (CA INDEX NAME)

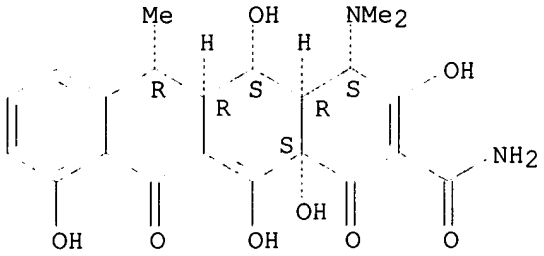


RN 139-85-5 HCAPLUS
CN Benzaldehyde, 3,4-dihydroxy- (9CI) (CA INDEX NAME)



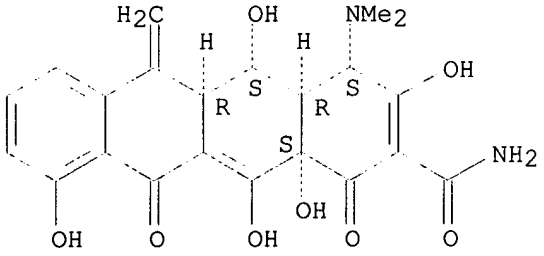
RN 564-25-0 HCAPLUS
CN 2-Naphthacenecarboxamide, 4-(dimethylamino)-1,4,4a,5,5a,6,11,12a-octahydro-3,5,10,12,12a-pentahydroxy-6-methyl-1,11-dioxo-, (4S,4aR,5S,5aR,6R,12aS)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



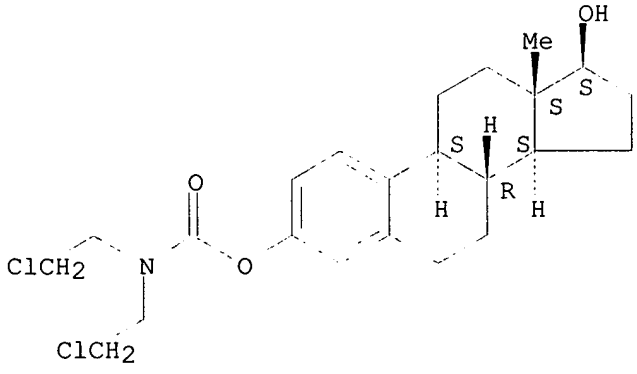
RN 914-00-1 HCAPLUS
CN 2-Naphthacenecarboxamide, 4-(dimethylamino)-1,4,4a,5,5a,6,11,12a-octahydro-3,5,10,12,12a-pentahydroxy-6-methylene-1,11-dioxo-, (4S,4aR,5S,5aR,12aS)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



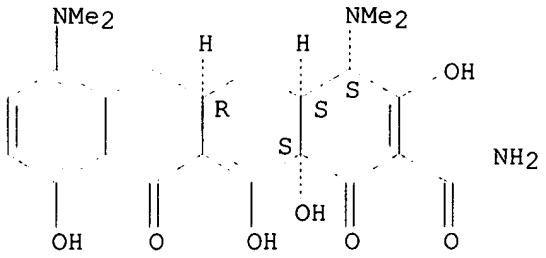
RN 2998-57-4 HCAPLUS
CN Estra-1,3,5(10)-triene-3,17-diol (17.beta.)-, 3-[bis(2-chloroethyl)carbamate] (9CI) (CA INDEX NAME)

Absolute stereochemistry.



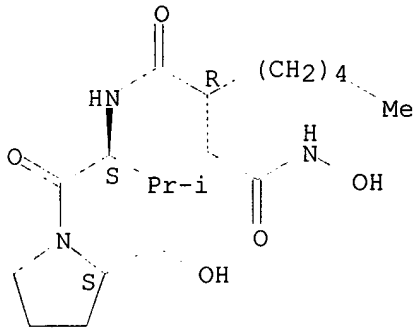
RN 10118-90-8 HCAPLUS
CN 2-Naphthacenecarboxamide, 4,7-bis(dimethylamino)-1,4,4a,5,5a,6,11,12a-octahydro-3,10,12,12a-tetrahydroxy-1,11-dioxo-, (4S,4aS,5aR,12aS)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



RN 13434-13-4 HCAPLUS
CN Butanediamide, N4-hydroxy-N1-[(1S)-1-[[(2S)-2-(hydroxymethyl)-1-pyrrolidinyl]carbonyl]-2-methylpropyl]-2-pentyl-, (2R)- (9CI) (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



RN 25378-27-2 HCAPLUS
CN Eicosapentaenoic acid (8CI, 9CI) (CA INDEX NAME)

CM 1

CRN 506-30-9
CMF C20 H40 O2

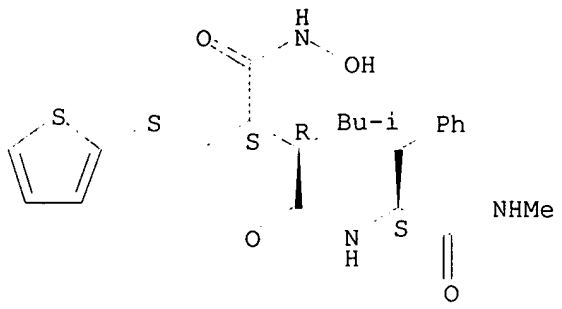
$HO_2C-(CH_2)_{18}-Me$

RN 51036-13-6 HCAPLUS
CN Ethanesulfonic acid, 2-(chloroamino)- (9CI) (CA INDEX NAME)

$ClNH-CH_2-CH_2-SO_3H$

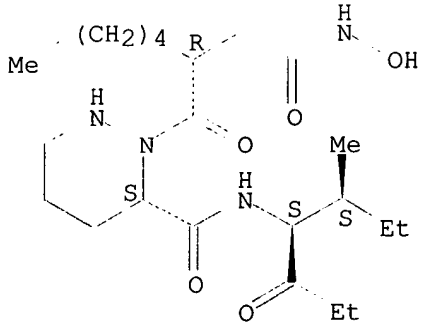
RN 130370-60-4 HCAPLUS
CN Butanediamide, N4-hydroxy-N1-[(1S)-2-(methylamino)-2-oxo-1-(phenylmethyl)ethyl]-2-(2-methylpropyl)-3-[(2-thienylthio)methyl]-, (2R,3S)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



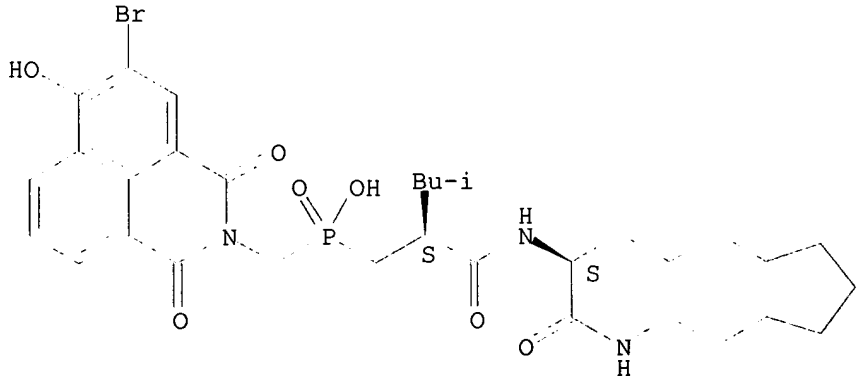
RN 140923-32-6 HCAPLUS
CN 1(2H)-Pyridazinebutanamide, tetrahydro-N-hydroxy-6-[[[(1S,2S)-2-methyl-1-(1-oxopropyl)butyl]amino]carbonyl]-.gamma.-oxo-.beta.-pentyl-, (.beta.R,6S)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



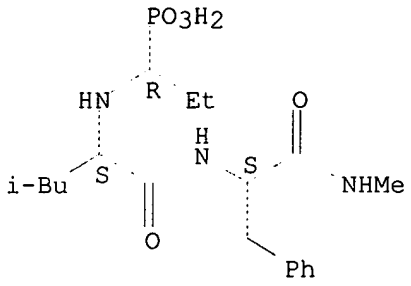
RN 141368-50-5 HCAPLUS
CN Phosphinic acid, [(5-bromo-6-hydroxy-1,3-dioxo-1H-benz[de]isoquinolin-2(3H)-yl)methyl][(2S)-4-methyl-2-[[[(3S)-2-oxoazacyclotridec-3-yl]amino]carbonyl]pentyl]- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



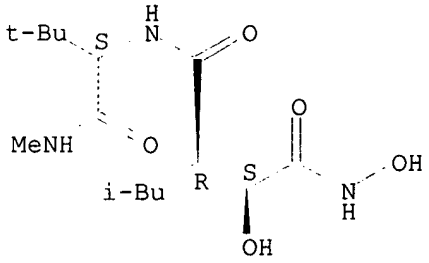
RN 153743-26-1 HCAPLUS
CN L-Phenylalaninamide, N-(1-phosphonopropyl)-L-leucyl-N-methyl-, (R)-(9CI) (CA INDEX NAME)

Absolute stereochemistry.



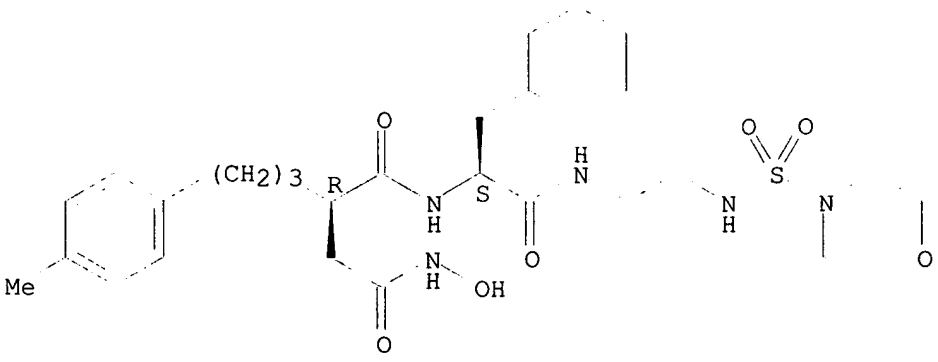
RN 154039-60-8 HCAPLUS
CN Butanediamide, N4-[(1S)-2,2-dimethyl-1-[(methylamino)carbonyl]propyl]-N1,2-dihydroxy-3-(2-methylpropyl)-, (2S,3R)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



RN 157549-53-6 HCAPLUS
CN Butanediamide, N1-[(1S)-1-(cyclohexylmethyl)-2-[[2-[(4-morpholinylsulfonyl)amino]ethyl]amino]-2-oxoethyl]-N4-hydroxy-2-[3-(4-methylphenyl)propyl]-, (2R)- (9CI) (CA INDEX NAME)

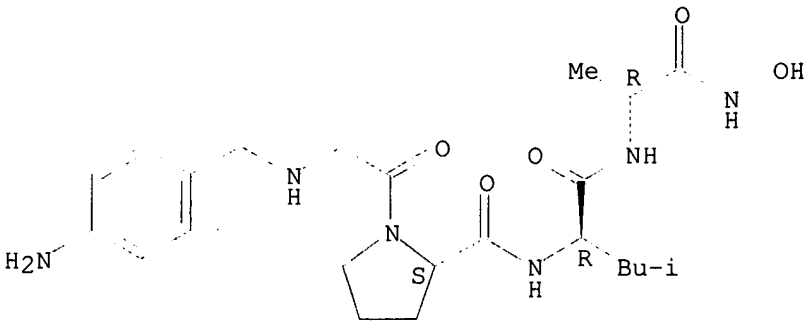
Absolute stereochemistry.



RN 209056-82-6 HCAPLUS

CN D-Alaninamide, N-[(4-aminophenyl)methyl]glycyl-L-prolyl-D-leucyl-N-hydroxy- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



IT **141907-41-7**, Matrix metalloproteinase **146480-35-5**, Matrix metalloproteinase-2 **146480-36-6**, Matrix metalloproteinase-9
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(matrix metalloproteinase inhibitors for redn. of unwanted hair growth)

RN 141907-41-7 HCAPLUS
CN Proteinase, matrix metallo- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

RN 146480-35-5 HCAPLUS
CN Gelatinase A (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

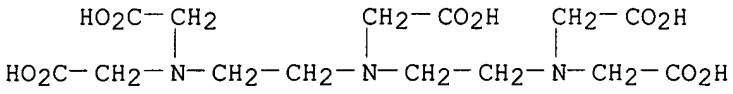
RN 146480-36-6 HCAPLUS
CN Gelatinase B (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

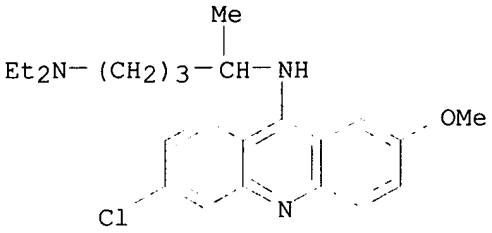
=> d bib abs hitstr 3

L5 ANSWER 3 OF 4 HCAPLUS COPYRIGHT 1998 ACS
AN 1996:660913 HCAPLUS
DN 125:293042
TI Use of angiogenesis suppressors for inhibiting hair growth
IN **Ahluwalia, Gurpreet S.; Styczynski, Peter;** Shander, Douglas
PA Handelman, Joseph H., USA
SO PCT Int. Appl., 23 pp.
CODEN: PIXXD2
PI WO 9626712 A2 19960906
DS W: AL, AM, AT, AU, AZ, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI
RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, DE, DK, ES, FR, GA, GB, GR, IE, IT, LU, MC, ML, NL, PT, SE
AI WO 96-US2790 19960227
PRAI US 95-396446 19950228
DT Patent
LA English
AB A method of inhibiting hair growth in a mammal includes applying, to an area of skin from which reduced hair growth is desired, a dermatol. acceptable compn. contg. a non-steroidal suppressor of angiogenesis. The effective compds. include sulfotransferase inhibitors, heparin binding antagonists, Cu chelators, histidine decarboxylase inhibitors, mast cell degranulation inhibitors, histamine receptor antagonists, ACE inhibitors, angiotensin II receptor antagonists, prostaglandin synthetase inhibitors, NK1 receptor antagonists, PAF receptor antagonists, and cytochrome P 450 reductase inhibitors. A topical prepn. contg. 10 % bathocuproine, was applied to male intact Golden Syrian hamsters; hair growth was inhibited by 81 %.
IT **67-43-6**, Diethylenetriamine pentaacetic acid **83-89-6**, Quinacrine **91-81-6**, Tripelennamine **113-92-8** **120-80-9**, 1,2-Benzenediol, biological studies **1398-62-5**, Chitin sulfate **1845-11-0**, Nafoxidine **3316-09-4**, p-Nitrocatechol **4431-00-9**, Aurintricarboxylic acid **4733-39-5**, Bathocuproine **7491-74-9**, Piracetam **10540-29-1**, Tamoxifen **12772-57-5**, Radicicol **15826-37-6**, Cromoglycate **18550-55-5**, Hyponitric acid **21829-25-4**, Nifedipine **23110-15-8**, Fumagillin **23593-75-1**, Clotrimazole **24280-93-1**, Mycophenolic acid **25614-03-3**, Bromocryptine **37270-94-3**, Platelet factor-4 **38096-31-0D**, Diaminoanthraquinone, derivs. **50679-08-8**, Terfenadine **51481-61-9**, Cimetidine **52698-84-7**, Bathocuproinesulfonate **57381-26-7**, Irsogladine **65899-73-2**, Tioconazole **70050-43-0**, .alpha.-Fluoromethylhistidine **75847-73-3**, Enalapril **76547-98-3**, Lisinopril **84088-42-6**, Linomide **110590-61-9** **114798-26-4**, Losartan **126509-46-4**, Eponemycin **129912-34-1** **135911-02-3** **182930-58-1**
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(angiogenesis suppressors for inhibiting hair growth)

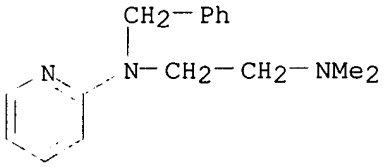
RN 67-43-6 HCAPLUS
CN Glycine, N,N-bis[2-[bis(carboxymethyl)amino]ethyl]- (7CI, 8CI, 9CI) (CA INDEX NAME)



RN 83-89-6 HCAPLUS
CN 1,4-Pentanediamine, N4-(6-chloro-2-methoxy-9-acridinyl)-N1,N1-diethyl- (9CI) (CA INDEX NAME)



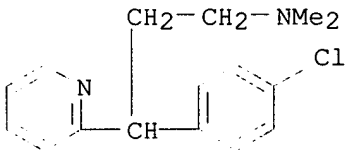
RN 91-81-6 HCAPLUS
CN 1,2-Ethanediamine, N,N-dimethyl-N'-(phenylmethyl)-N'-2-pyridinyl- (9CI) (CA INDEX NAME)



RN 113-92-8 HCAPLUS
CN 2-Pyridinepropanamine, .gamma.-(4-chlorophenyl)-N,N-dimethyl-, (2Z)-2-butenedioate (1:1) (9CI) (CA INDEX NAME)

CM 1

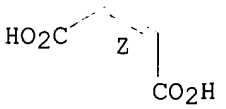
CRN 132-22-9
CMF C16 H19 Cl N2



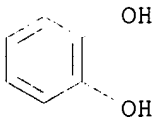
CM 2

CRN 110-16-7
CMF C4 H4 O4
CDES 2:Z

Double bond geometry as shown.



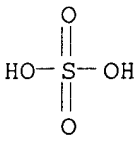
RN 120-80-9 HCAPLUS
CN 1,2-Benzenediol (9CI) (CA INDEX NAME)



RN 1398-62-5 HCAPLUS
CN Chitin, hydrogen sulfate (ester) (9CI) (CA INDEX NAME)

CM 1

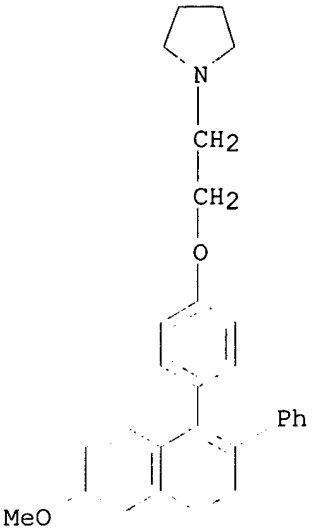
CRN 7664-93-9
CMF H2 O4 S



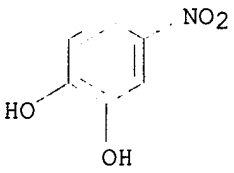
CM 2

CRN 1398-61-4
CMF Unspecified
CCI MAN

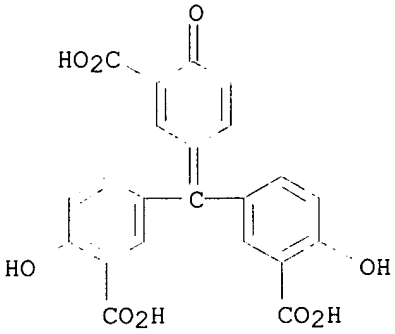
*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 1845-11-0 HCAPLUS
CN Pyrrolidine, 1-[2-[4-(3,4-dihydro-6-methoxy-2-phenyl-1-naphthalenyl)phenoxy]ethyl]- (9CI) (CA INDEX NAME)



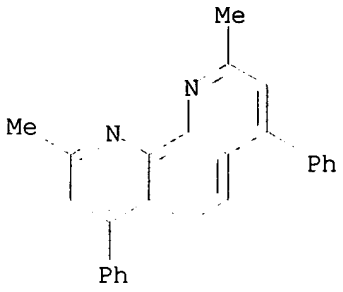
RN 3316-09-4 HCAPLUS
CN 1,2-Benzenediol, 4-nitro- (9CI) (CA INDEX NAME)



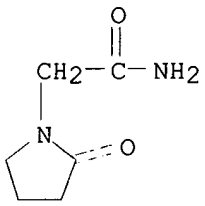
RN 4431-00-9 HCAPLUS
CN Benzoic acid, 5-[(3-carboxy-4-hydroxyphenyl)(3-carboxy-4-oxo-2,5-cyclohexadien-1-ylidene)methyl]-2-hydroxy- (9CI) (CA INDEX NAME)



RN 4733-39-5 HCAPLUS
CN 1,10-Phenanthroline, 2,9-dimethyl-4,7-diphenyl- (6CI, 7CI, 8CI, 9CI) (CA INDEX NAME)

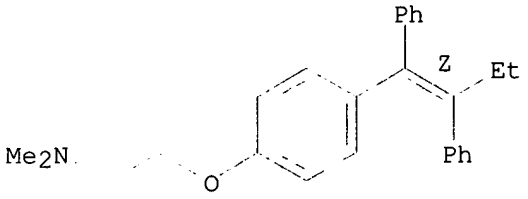


RN 7491-74-9 HCAPLUS
CN 1-Pyrrolidineacetamide, 2-oxo- (7CI, 8CI, 9CI) (CA INDEX NAME)

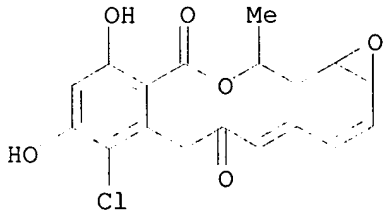


RN 10540-29-1 HCAPLUS
CN Ethanamine, 2-[4-[(1Z)-1,2-diphenyl-1-butenyl]phenoxy]-N,N-dimethyl- (9CI) (CA INDEX NAME)

Double bond geometry as shown.

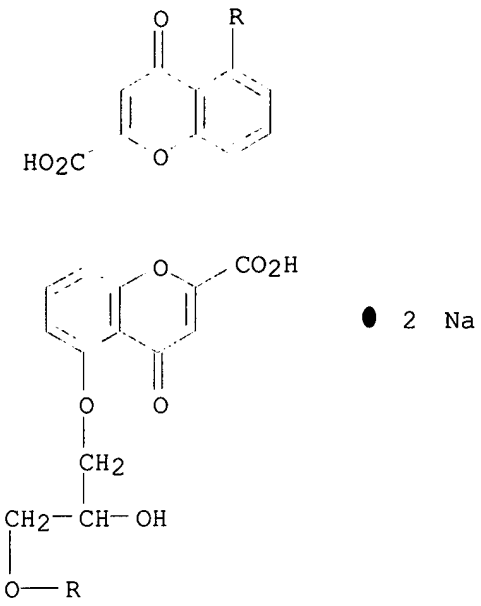


RN 12772-57-5 HCAPLUS
CN 6H-Oxireno[e][2]benzoxacyclotetradecin-6,12(7H)-dione, 8-chloro-1a,14,15,15a-tetrahydro-9,11-dihydroxy-14-methyl-, (1aS,2Z,4E,14R,15aS)- (9CI) (CA INDEX NAME)

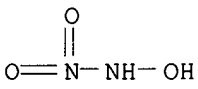


RN 15826-37-6 HCAPLUS
CN 4H-1-Benzopyran-2-carboxylic acid, 5,5'-[(2-hydroxy-1,3-

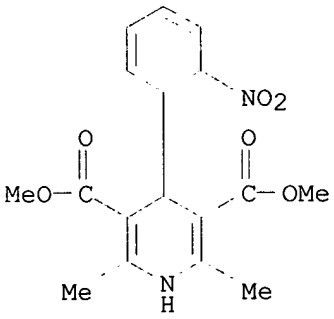
propanediyl)bis(oxy)]bis[4-oxo-, disodium salt (9CI) (CA INDEX NAME)



RN 18550-55-5 HCAPLUS
CN Hyponitric acid (6CI, 8CI, 9CI) (CA INDEX NAME)

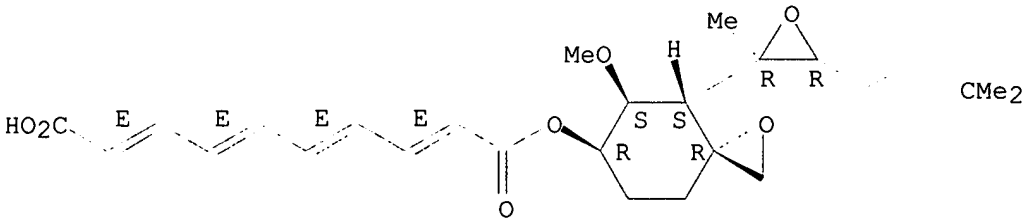


RN 21829-25-4 HCAPLUS
CN 3,5-Pyridinedicarboxylic acid, 1,4-dihydro-2,6-dimethyl-4-(2-nitrophenyl)-, dimethyl ester (9CI) (CA INDEX NAME)

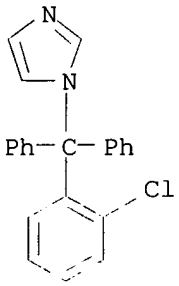


RN 23110-15-8 HCAPLUS
CN 2,4,6,8-Decatetraenedioic acid, mono[(3R,4S,5S,6R)-5-methoxy-4-[(2R,3R)-2-methyl-3-(3-methyl-2-butenyl)oxiranyl]-1-oxaspiro[2.5]oct-6-yl] ester, (2E,4E,6E,8E)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.
Double bond geometry as shown.

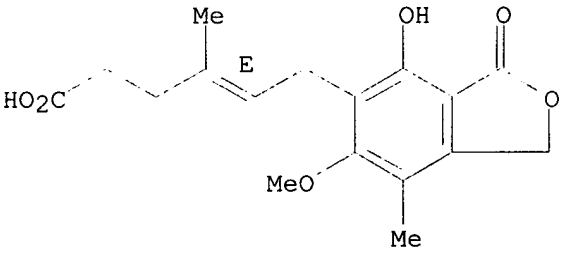


RN 23593-75-1 HCAPLUS
CN 1H-Imidazole, 1-[(2-chlorophenyl)diphenylmethyl]- (9CI) (CA INDEX NAME)



RN 24280-93-1 HCAPLUS
CN 4-Hexenoic acid, 6-(1,3-dihydro-4-hydroxy-6-methoxy-7-methyl-3-oxo-5-isobenzofuranyl)-4-methyl-, (4E)- (9CI) (CA INDEX NAME)

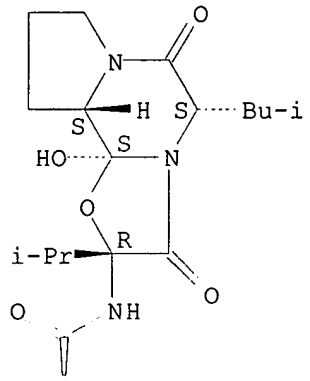
Double bond geometry as shown.



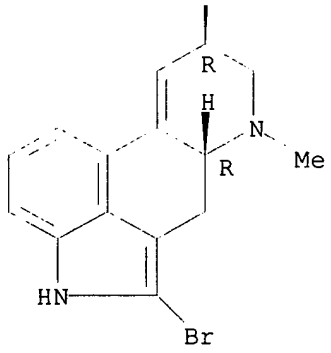
RN 25614-03-3 HCAPLUS
CN Ergotaman-3',6',18-trione, 2-bromo-12'-hydroxy-2'-(1-methylethyl)-5'-(2-methylpropyl)-, (5'.alpha.)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A

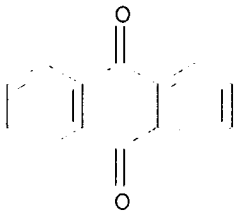


PAGE 2-A



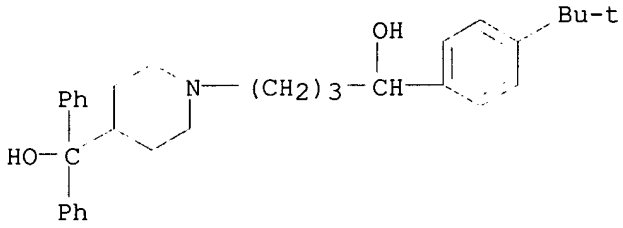
RN 37270-94-3 HCAPLUS
CN Blood platelet factor 4 (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 38096-31-0 HCAPLUS
CN 9,10-Anthracenedione, diamino- (9CI) (CA INDEX NAME)

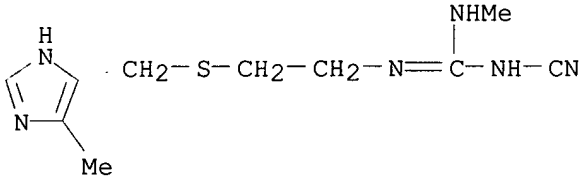


2 [D1—NH_2]

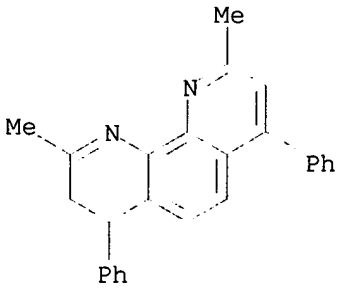
RN 50679-08-8 HCAPLUS
CN 1-Piperidinebutanol, .alpha.-[4-(1,1-dimethylethyl)phenyl]-4-(hydroxydiphenylmethyl)- (9CI) (CA INDEX NAME)



RN 51481-61-9 HCAPLUS
CN Guanidine, N-cyano-N'-methyl-N''-[2-[[(5-methyl-1H-imidazol-4-yl)methyl]thio]ethyl]- (9CI) (CA INDEX NAME)



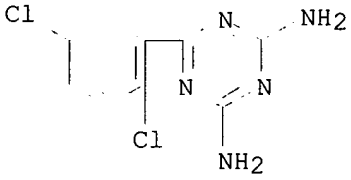
RN 52698-84-7 HCAPLUS
CN 1,10-Phenanthroline, 2,9-dimethyl-4,7-diphenyl-, disulfo deriv., disodium salt (9CI) (CA INDEX NAME)



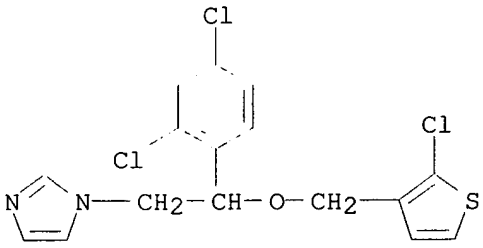
2 [D1—SO_3H]

● 2 Na

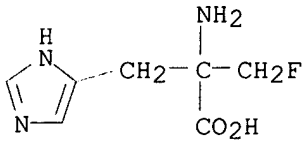
RN 57381-26-7 HCAPLUS
CN 1,3,5-Triazine-2,4-diamine, 6-(2,5-dichlorophenyl)- (9CI) (CA INDEX NAME)



RN 65899-73-2 HCAPLUS
CN 1H-Imidazole, 1-[2-[(2-chloro-3-thienyl)methoxy]-2-(2,4-dichlorophenyl)ethyl]- (9CI) (CA INDEX NAME)

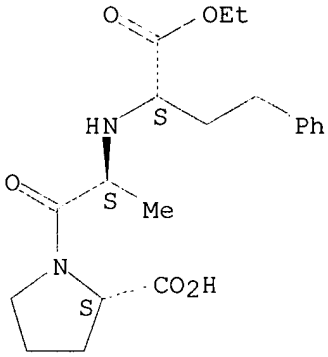


RN 70050-43-0 HCAPLUS
CN Histidine, .alpha.-(fluoromethyl)- (9CI) (CA INDEX NAME)



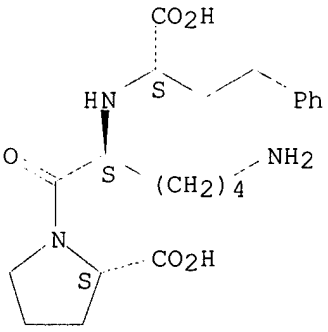
RN 75847-73-3 HCAPLUS
CN L-Proline, N-[(1S)-1-(ethoxycarbonyl)-3-phenylpropyl]-L-alanyl- (9CI) (CA INDEX NAME)

Absolute stereochemistry.

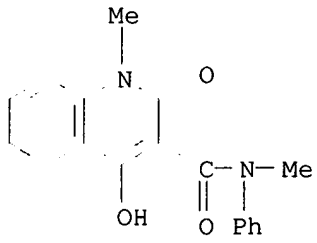


RN 76547-98-3 HCAPLUS
CN L-Proline, N2-[(1S)-1-carboxy-3-phenylpropyl]-L-lysyl- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



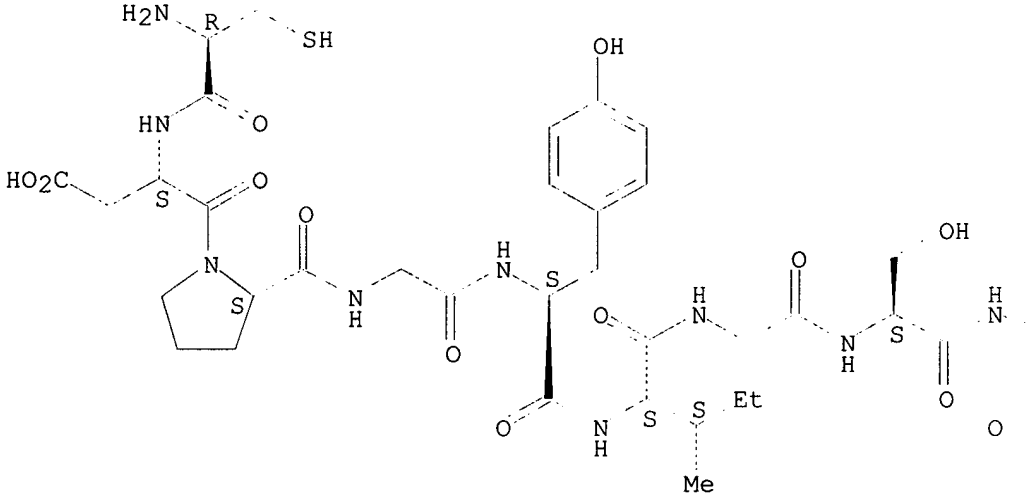
RN 84088-42-6 HCAPLUS
CN 3-Quinolinecarboxamide, 1,2-dihydro-4-hydroxy-N,1-dimethyl-2-oxo-N-phenyl- (9CI) (CA INDEX NAME)



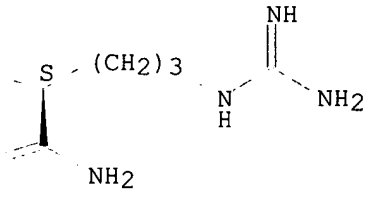
RN 110590-61-9 HCAPLUS
CN L-Argininamide, L-cysteinyl-L-.alpha.-aspartyl-L-prolylglycyl-L-tyrosyl-L-isoleucylglycyl-L-seryl- (9CI) (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).

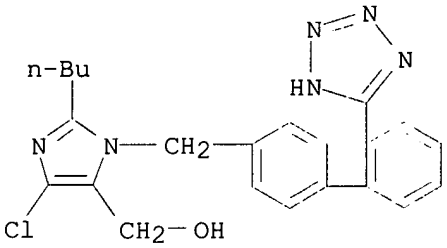
PAGE 1-A



PAGE 1-B

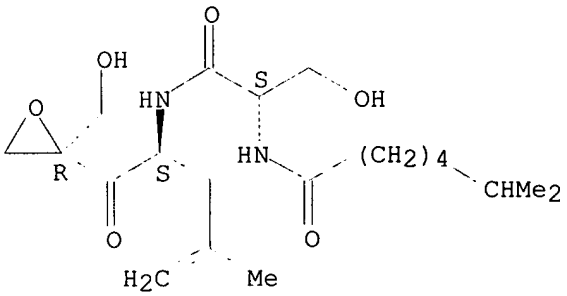


RN 114798-26-4 HCAPLUS
CN 1H-Imidazole-5-methanol, 2-butyl-4-chloro-1-[[2'-(1H-tetrazol-5-yl)[1,1'-biphenyl]-4-yl]methyl]- (9CI) (CA INDEX NAME)



RN 126509-46-4 HCAPLUS
CN Heptanamide, N-[1-(hydroxymethyl)-2-[[1-[[2-(hydroxymethyl)oxiranyl]carbonyl]-3-methyl-3-butenyl]amino]-2-oxoethyl]-6-methyl-, [2R-[2R*[S*(S*)]]]- (9CI) (CA INDEX NAME)

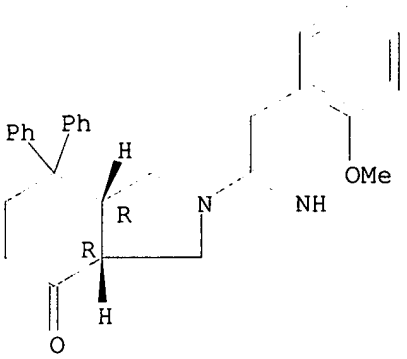
Absolute stereochemistry.



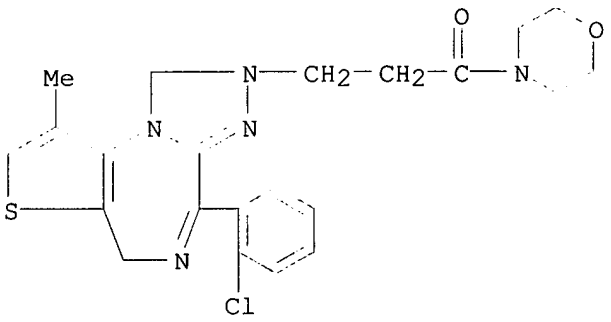
RN 129912-34-1 HCAPLUS
RN 135911-02-3 HCAPLUS

CN 4H-Isoindol-4-one, octahydro-2-[1-imino-2-(2-methoxyphenyl)ethyl]-7,7-diphenyl-, (3aR,7aR)- (9CI) (CA INDEX NAME)

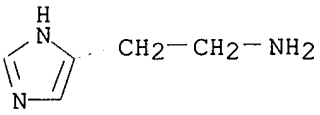
Absolute stereochemistry.



RN 182930-58-1 HCAPLUS
CN Morpholine, 4-[3-[4-(2-chlorophenyl)-9-methyl-1H-thieno[2,3-f][1,2,4]triazolo[4,3-a][1,4]diazepin-2(6H)-yl]-1-oxopropyl]- (9CI) (CA INDEX NAME)



IT **51-45-6**, Histamine, biological studies **11128-99-7**, Angiotensin II **33507-63-0**, Substance P **65154-06-5**, Platelet activating factor
RL: BSU (Biological study, unclassified); BIOL (Biological study) (antagonists; angiogenesis suppressors for inhibiting hair growth)
RN 51-45-6 HCAPLUS
CN 1H-Imidazole-4-ethanamine (9CI) (CA INDEX NAME)



RN 11128-99-7 HCAPLUS
CN Angiotensin II (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

RN 33507-63-0 HCAPLUS
CN Substance P (9CI) (CA INDEX NAME)

Absolute stereochemistry.

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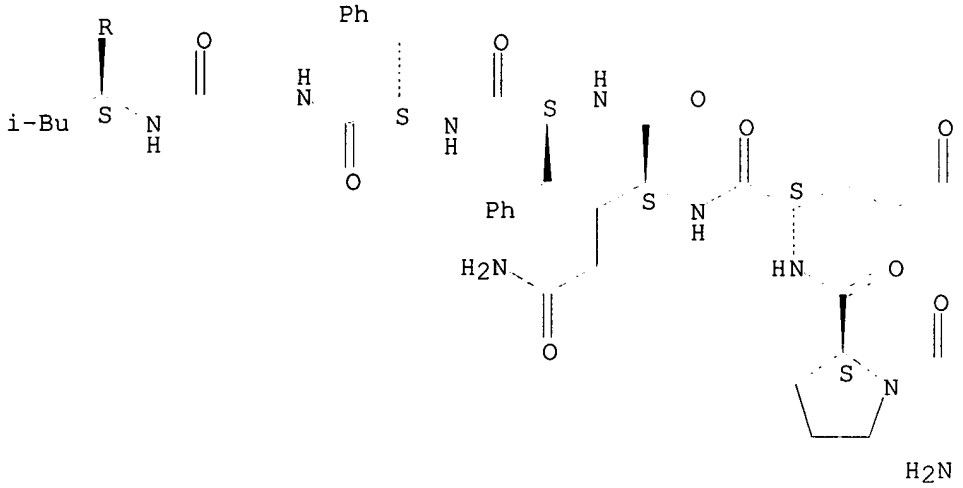
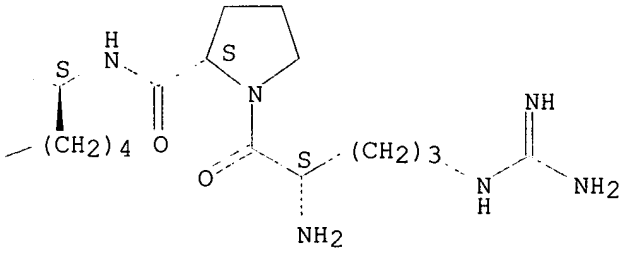
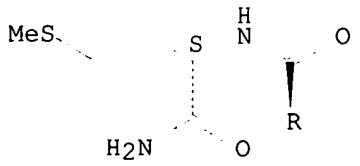


PAGE 1-B



PAGE 2-A



RN 65154-06-5 HCAPLUS
CN Blood platelet-activating factor (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
IT **9015-82-1**, Angiotensin-converting enzyme **9023-09-0**
, Sulfotransferase **9024-61-7**, Histidine decarboxylase
9039-06-9, Cytochrome P450 reductase **9055-65-6**,
Prostaglandin synthetase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(inhibitors; angiogenesis suppressors for inhibiting hair growth)
RN 9015-82-1 HCAPLUS
CN Carboxypeptidase, dipeptidyl (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 9023-09-0 HCAPLUS
CN Sulfotransferase (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 9024-61-7 HCAPLUS
CN Decarboxylase, histidine (9CI) (CA INDEX NAME)

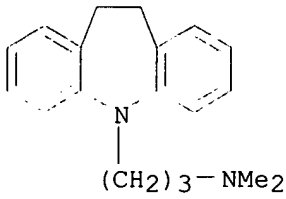
*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 9039-06-9 HCAPLUS
CN Reductase, cytochrome P 450 (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 9055-65-6 HCAPLUS
CN Synthase, prostaglandin (9CI) (CA INDEX NAME)

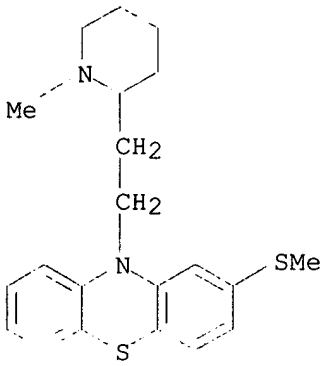
*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

=> d bib abs hitstr 4

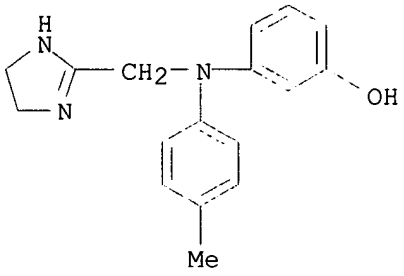
L5 ANSWER 4 OF 4 HCAPLUS COPYRIGHT 1998 ACS
AN 1996:354097 HCAPLUS
DN 125:18662
TI Inhibition of hair growth with protein kinase C inhibitors
IN **Ahluwalia, Gurpreet S.**; Shander, Douglas; **Styczynski, Peter**
PA Handelman, Joseph, H., USA
SO PCT Int. Appl., 14 pp.
CODEN: PIXXD2
PI WO 9609806 A2 19960404
DS W: AM, AT, AU, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM
RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, DE, DK, ES, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG
AI WO 95-US12134 19950921
PRAI US 94-314327 19940928
DT Patent
LA English
AB Mammalian hair growth is reduced by applying to the skin a compn. including an inhibitor of protein kinase C (PKC). The inhibitor interacts with the ATP-binding site, Ca-binding site, or phospholipid-interacting site in PKC. The compn. provides a redn. in hair growth of .gtoreq.30% when tested in the Golden Syrian hamster assay. A no. of PKC inhibitors were tested in the Golden Syrian hamster assay; e.g. verapamil, thioridazine, curcumin, and trifluoperazine inhibited hair growth by 56-69%.
IT **50-49-7**, Imipramine **50-52-2**, Thioridazine **50-60-2**, Phentolamine **52-53-9**, Verapamil **92-84-2D**, Phenothiazine, derivs. **117-89-5**, Trifluoperazine **137-66-6**, Ascorbic acid 6-palmitate **458-37-7**, Curcumin **471-53-4**, 18.beta.-Glycyrrhetinic acid **1404-26-8**, Polymyxin B **1405-86-3**, Glycyrrhetinic acid glycoside **6707-58-0**, Dequalinium **18417-89-5**, Sangivamycin **22990-77-8**, 2-(Aminomethyl)piperidine **23214-92-8D**, Doxorubicin, iron complexes **62996-74-1D**, Staurosporine, derivs. **63590-19-2**, Balanol **84477-87-2**, 1-(5-Isoquinolinylsulfonyl)-2-methylpiperazine **100107-43-5D**, Isoquinolinesulfonamide, derivs. **110124-55-5** **133052-90-1**, GF 109203X
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(hair growth inhibition with protein kinase C inhibitors)
RN 50-49-7 HCAPLUS
CN 5H-Dibenz[b,f]azepine-5-propanamine, 10,11-dihydro-N,N-dimethyl- (9CI) (CA INDEX NAME)



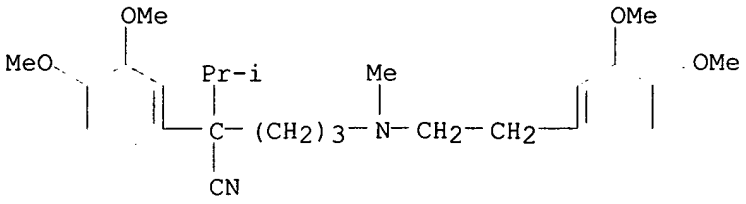
RN 50-52-2 HCAPLUS
CN 10H-Phenothiazine, 10-[2-(1-methyl-2-piperidinyl)ethyl]-2-(methylthio)- (9CI) (CA INDEX NAME)



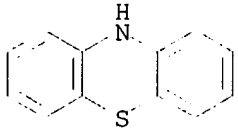
RN 50-60-2 HCAPLUS
CN Phenol, 3-[[(4,5-dihydro-1H-imidazol-2-yl)methyl](4-methylphenyl)amino]- (9CI) (CA INDEX NAME)



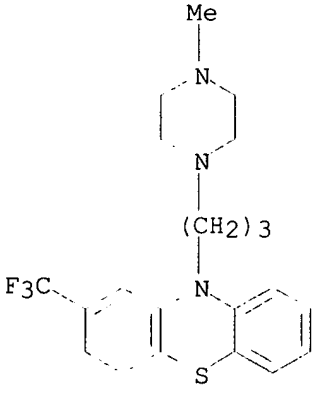
RN 52-53-9 HCAPLUS
CN Benzeneacetonitrile, .alpha.-[3-[[2-(3,4-dimethoxyphenyl)ethyl]methylamino]propyl]-3,4-dimethoxy-.alpha.-(1-methylethyl)- (9CI) (CA INDEX NAME)



RN 92-84-2 HCAPLUS
CN 10H-Phenothiazine (9CI) (CA INDEX NAME)

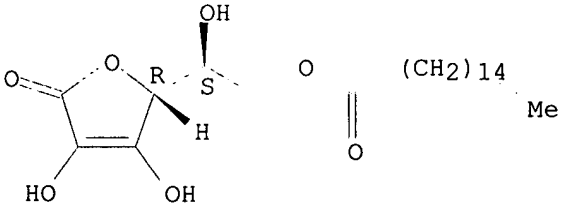


RN 117-89-5 HCAPLUS
CN 10H-Phenothiazine, 10-[3-(4-methyl-1-piperazinyl)propyl]-2-(trifluoromethyl)- (9CI) (CA INDEX NAME)



RN 137-66-6 HCAPLUS
CN L-Ascorbic acid, 6-hexadecanoate (9CI) (CA INDEX NAME)

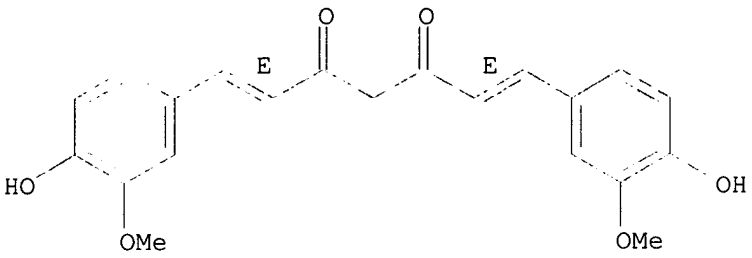
Absolute stereochemistry.



RN 458-37-7 HCAPLUS
CN 1,6-Heptadiene-3,5-dione, 1,7-bis(4-hydroxy-3-methoxyphenyl)-,

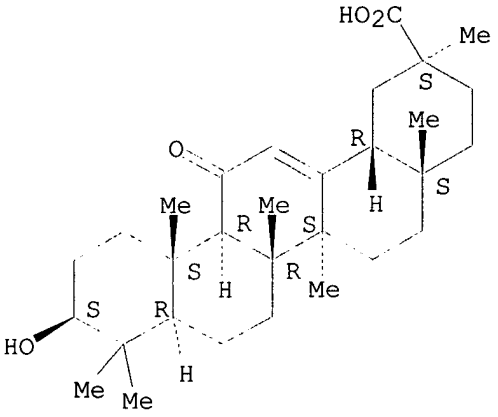
(E,E)- (8CI, 9CI) (CA INDEX NAME)

Double bond geometry as shown.



RN 471-53-4 HCAPLUS
CN Olean-12-en-29-oic acid, 3-hydroxy-11-oxo-, (3.beta.,20.beta.)- (9CI) (CA INDEX NAME)

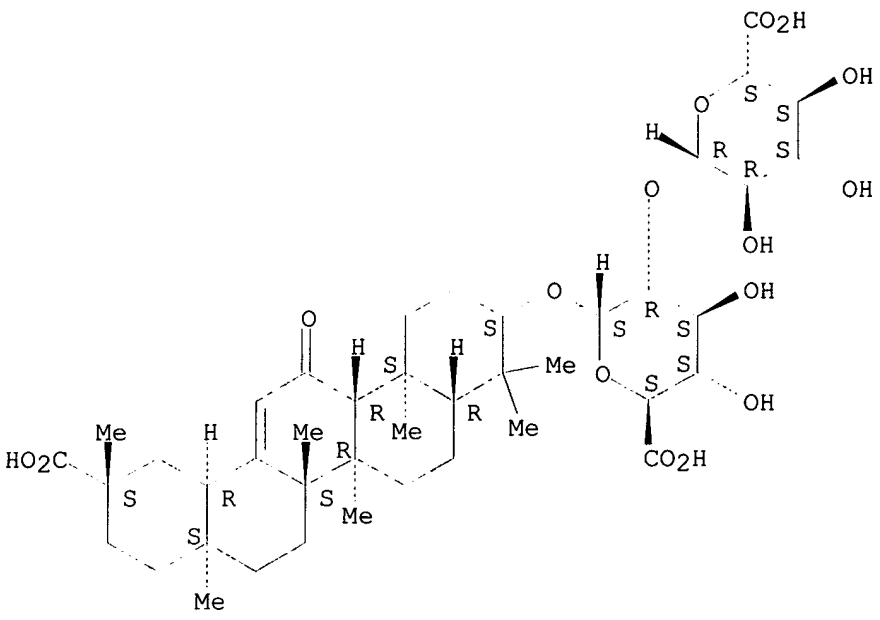
Absolute stereochemistry.



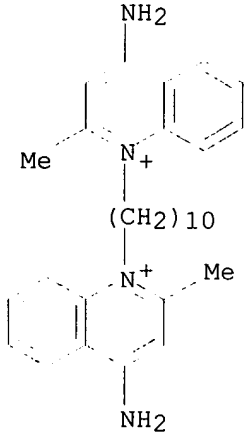
RN 1404-26-8 HCAPLUS
CN Polymyxin B (7CI, 8CI, 9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 1405-86-3 HCAPLUS
CN .alpha.-D-Glucopyranosiduronic acid, (3.beta.,20.beta.)-20-carboxy-11-oxo-30-norolean-12-en-3-yl 2-O-.beta.-D-glucopyranuronosyl- (9CI) (CA INDEX NAME)

Absolute stereochemistry.

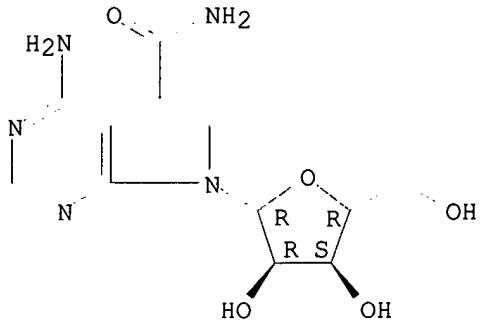


RN 6707-58-0 HCAPLUS
CN Quinolinium, 1,1'-(1,10-decanediyl)bis[4-amino-2-methyl- (9CI) (CA INDEX NAME)

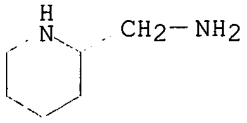


RN 18417-89-5 HCAPLUS
CN 7H-Pyrrolo[2,3-d]pyrimidine-5-carboxamide, 4-amino-7-.beta.-D-ribofuranosyl- (8CI, 9CI) (CA INDEX NAME)

Absolute stereochemistry.

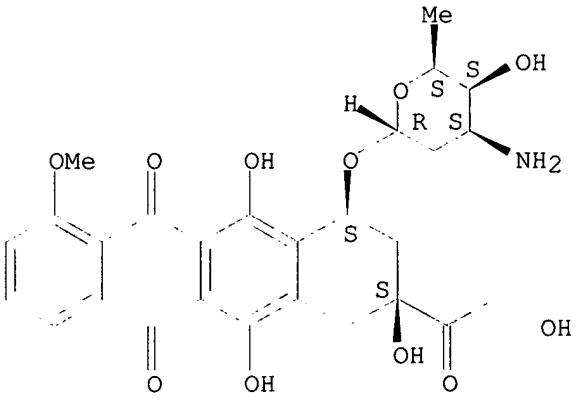


RN 22990-77-8 HCAPLUS
CN 2-Piperidinemethanamine (9CI) (CA INDEX NAME)



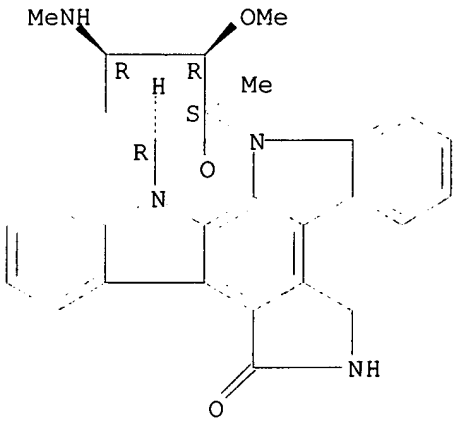
RN 23214-92-8 HCAPLUS
CN 5,12-Naphthacenedione, 10-[(3-amino-2,3,6-trideoxy-.alpha.-L-lyxo-hexopyranosyl)oxy]-7,8,9,10-tetrahydro-6,8,11-trihydroxy-8-(hydroxyacetyl)-1-methoxy-, (8S,10S)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



RN 62996-74-1 HCAPLUS
CN 9,13-Epoxy-1H,9H-diindolo[1,2,3-gh:3',2',1'-lm]pyrrolo[3,4-j][1,7]benzodiazonin-1-one, 2,3,10,11,12,13-hexahydro-10-methoxy-9-methyl-11-(methylamino)-, (9S,10R,11R,13R)- (9CI) (CA INDEX NAME)

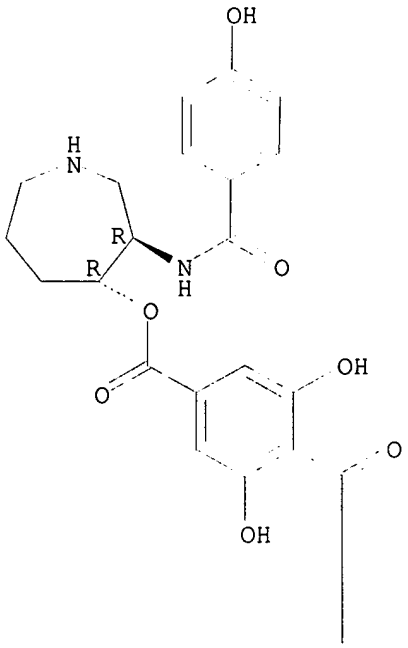
Absolute stereochemistry. Rotation (+).



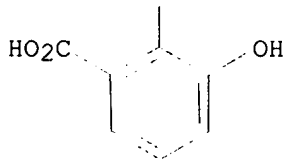
RN 63590-19-2 HCAPLUS
CN Benzoic acid, 4-(2-carboxy-6-hydroxybenzoyl)-3,5-dihydroxy-, 1-[(3R,4R)-hexahydro-3-[(4-hydroxybenzoyl)amino]-1H-azepin-4-yl] ester (9CI) (CA INDEX NAME)

Absolute stereochemistry.

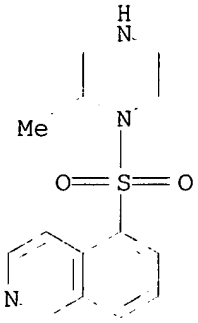
PAGE 1-A



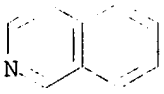
PAGE 2-A

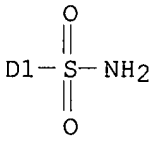


RN 84477-87-2 HCAPLUS
CN Piperazine, 1-(5-isoquinolinylsulfonyl)-2-methyl- (9CI) (CA INDEX NAME)

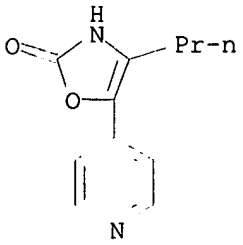


RN 100107-43-5 HCAPLUS
CN Isoquinolinesulfonamide (9CI) (CA INDEX NAME)

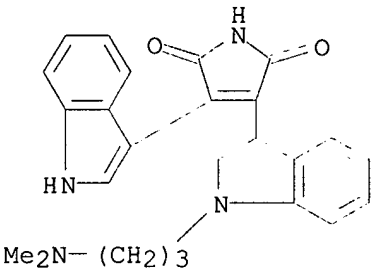




RN 110124-55-5 HCAPLUS
CN 2(3H)-Oxazolone, 4-propyl-5-(4-pyridinyl)- (9CI) (CA INDEX NAME)



RN 133052-90-1 HCAPLUS
CN 1H-Pyrrole-2,5-dione, 3-[1-[3-(dimethylamino)propyl]-1H-indol-3-yl]-4-(1H-indol-3-yl)- (9CI) (CA INDEX NAME)



IT **141436-78-4**, Protein kinase C
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(inhibitors; hair growth inhibition with protein kinase C inhibitors)
RN 141436-78-4 HCAPLUS
CN Kinase (phosphorylating), protein, C (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***